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Civil Defense in Texas

In this day of power-backed, all-out diplomatic effort among the nations of the world, many governments are threatened by unrest.

The government of Turkey was changed by use of military power. The government of Cuba was changed by a civil war. The government of South Korea has come into new hands through the use of force. The Japanese government was unable to prevent mass insult of our President. What country will lose its form of government next?

Will the next change start a big war? Red China plans to detonate an atomic device soon. Will Castro's Cuba be next in line to carry an atomic "six-gun," and start practicing the fast draw?

As physicians we must review our preparedness program. A few short years ago, there was no warehouse stock of medical and surgical instruments and supplies. Orders of large size had to be made as ordered in factories centered within a "bomb's throw" of Washington, D. C. Military strength had to be shored up. It became obvious to medical and governmental leaders of our country that medical equipment and supplies had to be built up to meet minimum needs of civilians in the event of atomic war. Once the critically short medical and surgical instrument supply was stockpiled in a few warehouses of the nation, problems of their protection from bombs, and of rapid distribution in time of need, became apparent. The solution came when 200 bed civil defense emergency hospitals were offered to cities and counties for storage near possible target areas.

In Texas, the assurance of hospital facilities with which our physicians can work, did not materialize until the physi-

cians themselves assumed aggressive roles in local areas all over Texas to make the prepositioning program one of the most successful in the nation.

There are today 69 emergency type hospitals in storage in "polka dot fashion" over our state and several are yet to be delivered. Most of our component societies have emergency medical service organizations. The organizations, and the staffing patterns, must be reviewed from time to time with a view of improving the volume and quality of service to be offered patients in time of war disaster. The few societies without plans for organized effort must face realities and develop plans in case there should be need for mass emergency care of burns, trauma, or radiation sickness in addition to the usual case-load of sick people under trying conditions.

In addition to preparing and planning curative measures for a possible disaster, we must practice good citizenship and watch for any developments within our communities which point to possible radical change in the form of our government. We must watch the group action of our college age people. It has been mob action by our young people in other countries which has precipitated dramatic changes in governments. These impressionable young people have had leadership. We must study movements, and search out and examine the leaders behind any mass alteration in the behavior of our student age sons and daughters.

—T. E. DODD, M.D., Austin, Chairman, Committee on Emergency Medical Service, Texas Medical Association.

The Torch of Hope

Only 12 short years have passed since a relatively small group of men and women, imbued with hope, crystallized the fight against heart disease into a single entity of unique force and effectiveness. I refer, of course, to the transformation of the American Heart Association from a small professional society into a national voluntary health agency of laymen, scientists, and physicians. At the same time, the Texas Heart Association became the state's official affiliate of the American Heart Association—the only agency devoted exclusively to the conquest of heart and circulatory ailments.

The year 1948 marked not only the beginning of the American Heart Association, but the actual beginning of the first planned and concerted research assault upon the heart diseases. At that time, we left behind an era in which heart research was restricted by lack of funds and competent investigators. We entered an era in which vastly enlarged research allocations, provided by public contributions, were available to support highly qualified medical scientists. We also entered an era in which totally new concepts of research support came into being, and, for the first time, attention was given to developing and training scientific manpower for cardiovascular research in the future.

In the past 12 years, many hopes have matured into realities with the investment of more than 50,000,000 Heart dollars on the state and national level in research. Here in Texas, the research program of the Texas Heart Association has grown at a phenomenally accelerated pace in the past 2 years—from $4,600 in 1958-1959 to requests totaling over $170,000 for the year beginning July 1, 1960. These requests came from 26 qualified research investigators located in six medical institutions in Texas. Physicians, by training, are well aware of the need for continued and expanded research in the cardiovascular field. It would be to the best interests of the medical profession and the people of Texas if more physicians took an even more active part in the work of the Texas Heart Association so that the research program, in particular, can be expanded even more in the months and years ahead.

Research, however, is only one of three major activities of your heart association. Heart dollars also have made it possible to carry forward educational programs—programs designed to

bring physicians new knowledge developed by the research scientist. The heart association gives the public accurate information about the heart diseases—information which helps to banish needless fears and to eliminate harmful misconceptions. And, it fights heart disease through community programs aimed at safeguarding healthy hearts and enabling sufferers from heart and circulatory ailments to retain their places in society as useful and productive citizens. More recently, it has taken the job of cardiovascular rehabilitation under its wing.

The heart-and-torch insignia should be viewed by physicians as a beacon of new hope for hearts everywhere for it is truly a symbol of confidence, determination, and faith.

—ROBERT E. LESLIE, M.D., El Campo,
President, Texas Heart Association.

Texas Doctors Deserve Praise

Although participation by Texas doctors in the American Medical Education Fund during 1959 still amounted to only 20 per cent, and there is room for continual improvement, the total increase in contributions was nearly 50 times higher than it was in 1952.

Physicians from Texas gave $50,113.25 last year while in 1952 they gave only $1,065. At that time participation was .32 per cent of the members of Texas Medical Association.

Donations have climbed rapidly, and Texas doctors deserve praise for their recognition of the merit of the educational fund. Three Texas medical schools currently are utilizing contributions made in 1959. Eighty-five medical schools were recipients recently of $1,198,334 from AMEF and Texas schools shared in $33,-151 of this. Portions went to Baylor University College of Medicine, Houston; the University of Texas Medical Branch, Galveston; and the University of Texas Southwestern Medical School, Dallas.

Here, at the core of medical research in our state, the money is being put to work to further the quality and standards of students, faculty, and research in medicine. All funds given through AMEF are sent directly to the deans of the medical schools to be used at their discretion and do not go into administrative expenses, which are paid by the American Medical Association. All funds do not necessarily return to Texas, so if your favorite school is out of state you may earmark your contribution for it.

It has been suggested frequently in recent months that the profession is losing ground at the student level—bright students are being wooed by representatives of other highly competitive scientific and professional fields. But despite this competition, medicine must progress, must meet the demands of the nation's people, and to do so it must have more and better students. Optimism is high, however, as evidenced by the great increase in participation and donations since the beginning years of AMEF. By giving between now and December 31, an even great amount of funds can be presented to the profession's own marketplace of learning and research—its medical schools.

Tumor Seminar

Our thanks to the San Antonio Society of Pathologists for its 1958 Tumor Seminar appearing in the original article section of this issue. Guest editor of the seminar is Lt. Col. James L. Hansen, chief of the Anatomical Pathology Section of Brooke Army Hospital, Fort Sam Houston, and now president of the San Antonio Society of Pathologists.

Col. George J. Matt, M.C., president of the society at the time of the seminar, opened the meeting. Dr. A. O. Severance, San Antonio, was moderator and Dr. William A. Meissner of New England Deaconess Hospital, Boston, was conductor of the seminar.

Sixteen cases are included in the seminar conducted by the New England pathologist.

President's Page

Wanted: Dedicated Students

DR. OWEN

We have a challenge: nation-wide statistics reveal that nearly half of our brightest high school students do not go on to college. This is potential leadership material lost.

Today—more than ever—when a higher premium is placed on excellence and on higher performance, we must continue to seek out thinkers and leaders. There is an urgent need to promote education that will improve human understanding and tolerance.

We cannot be content with our role in medical science in the hospitals and office. It is our duty to exert a greater influence in our communities as dedicated physicians.

Our county medical societies might do well to follow the example, or similar program of that set up by the Adams County Medical Society, Quincy, Ill., in 1956. They united with other civic organizations to encourage academic achievement. Their aim was to make college education and special training available for every talented high school graduate.

We should offer encouragement and, when necessary and possible, monetary aid to those who—without a helping hand—might not be able to secure the education necessary for a career in the medical field.

We are all aware that there is a pronounced decline in the number of applicants for medical schools. More outstanding is the decline of dedicated, or talented, men and women not only seeking medical training, but also allied medical vocations.

What action are we as an association, and individually, doing to meet the challenge?

The Nueces County Medical Society (Corpus Christi) has recently voted $1,000 to provide a scholarship each year to a deserving medical school student from that county. It is my understanding that, ultimately, the county society will have four boys or girls in school at the same time with one scholarship recipient in each class. This is a wonderful gesture—an example we would all do well to follow.

Our education program is a continuing one. In this connection, doctors are urged to avail themselves of the excellent program of the postgraduate medical assemblies and many others.

May Owen

Pro / Con

A column for readers' assents, dissents, and comments, selected monthly by the official advisory committee of the Texas State Journal of Medicine.

Internist Favors Drug Companies' Stand On Hearings Conducted by Senator Kefauver

The following letter was written by Dr. J. Edward Johnson, Austin internist, in response to a letter John E. McKeen, President of Charles Pfizer and Co., Inc., had directed to Senator Estes Kefauver. McKeen's letter concerned the writing of prescriptions for drugs by brand name instead of generic name. He endorsed the suggestion made by Dr. Alexander Marble of Boston, who testified before Kefauver's Committee, that a forum for scientific and objective review of the medical aspects of the oral antidiabetic drugs be held. McKeen believes that meetings of this type would allow the many specialists who prescribe such drugs to exchange experience in their use, and would assure the best possible therapeutic application of these agents.

Your letter of May 13 [Drs. Robert M. Rees and Robert C. Warner] to physicians protesting the methods and deploring the harm being done by the Kefauver committee hearings is appropriate and impressive. The letter of your President [Dr. McKeen] to Senator Kefauver suggests changes that could transform the Committee's investigation into useful service.

As a physician with more than 30 years' experience in the private practice of internal medicine, I am moved to comment. By further statement of background, I am chairman of the Committee on Scientific Exhibits of the Texas Medical Association, president of the Austin Heart Association, and president of the Texas Tuberculosis Association. In each of these capacities I am intensely interested in new drugs, their safety, and their usefulness.

For many years I have welcomed the drug "detail men" with their samples and literature. I learn much from them. As for the criticism that they wheedle, cajole, and bribe me to use their products, they do no such thing. The samples they leave are for clinical trial, and I am more than pleased to use them in selected cases in which I can supervise and control their administration. I do not accept them if I do not want them. The literature the firm sends or distributes via its representatives goes far to keep physicians abreast of recent advances.

Concerning the prescribing of drugs by generic instead of brand names, I do prefer generic names in

instances in which it is practical. Nevertheless, "Cantil" is a lot easier to write than "N-methyl-3-piperidyl-diphenyl glycolate methobromide!" The witness who states that doctors should prescribe by generic instead of brand name might be asked why he does not prescribe the kind of carburetor, oil pump, piston ring, transmission, etc.—all the way down to the tail pipe—that he wants on an automobile. There are companies who would put together such a vehicle for him. But no, he wants a Cadillac, Pontiac, or Rambler—and orders his car by brand name. In defense of the inconsistency, he might say, "I want a car with a reputation; I want to deal with a company I can trust."

The practicing physician is also a practical man, and he follows such a line of reasoning in regard to drugs. Furthermore, no other class of manufacturers can boast a better reputation for honest, above board dealing with the public than can the large pharmaceutical houses of America.

Physicians protest the meddling during these committee hearings with the doctor-patient relationship that is so important for the successful and efficient private practice of medicine. If objective investigations cannot be conducted by panels of competent scientists, it would be better to discontinue the hearings, as they produce more harm than good.

The antitrust laws should suffice to prevent conspiracy to avoid competition. If not, a more rational approach would be to amend the law, and then to direct attention of the Justice Department to presumed violations.

I am sure members of the medical profession will support the drug companies in an effort to direct the Committee into objective investigations and constructive procedure methods.

—J. EDWARD JOHNSON, M.D., Austin.

Letters are chosen on any subject on the basis of good taste, propriety, and available space by the Journal Advisory Committee. No anonymous letters will be printed and the committee reserves the right to print and to edit contributions. Letters do not necessarily represent the policy of the Texas Medical Association or the Journal.

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ORIGINAL ARTICLES

A Tumor Seminar

Proceedings of the fifteenth annual tumor seminar presented by the San Antonio Society of Pathologists at Brooke Army Hospital, Fort Sam Houston, on October 4, 1958. Conductor was Dr. William A. Meissner, Boston.

GUEST EDITOR:

LT. COL. JAMES L. HANSEN, Fort Sam Houston

Case 1 (Carotid Body Tumor)

Diagnosis.—Carotid body tumor (chemodectoma).

Contributor.—Col. J. M. Lukeman, M.C., Fourth United States Army Medical Laboratory, Fort Sam Houston.

History.—A 65 year old white female had a 5 year history of a very slowly enlarging mass in the left submandibular area with fluctuations in size from day to day. No calcification was seen by x-ray. The mass was firm, slightly tender, nonpulsatile, and fixed to deep structures. At surgery, an extremely vascular mass was found located at the bifurcation of the left carotid artery and densely adherent to both internal and external carotids. The mass was excised intact and was an encapsulated 3 by 2 by 2 cm., somewhat rubbery nodule with a brown to white to hemorrhagic cut surface.

DR. WILLIAM A. MEISSNER, Boston: The significant points in the history are the slow growth of the tumor, its location at the bifurcation of the carotid, and its extreme vascularity. From these points alone, the diagnosis is suggested strongly.

Microscopically, the tumor appears to be circumscribed. The stroma of the tumor is scanty, except in foci where the fibrous tissue appears to be reactive rather than a part of the neoplasm. Also prominent in the tumor is the great vascularity with vessels of varying size surrounded closely by tumor cells. The tumor cells are of fairly uniform large size and are epithelioid. The nuclei are round or ovoid, without mitoses. The cytoplasm is often rather clear,

which almost suggests that there might be contained material such as lipid or glycogen. The characteristic feature of the tumor cells is their accumulation into clusters surrounded by a fine prolongation of the trabecular fibrous tissue. The tendency of the cells to form clusters is seen more clearly with the silver stain. These features add up to the typical appearance of a carotid body tumor, which diagnosis can be made with little need of going into a differential.

Submitted Diagnoses.—tumor of carotid body (chemodectoma) 33; nonchromaffin paraganglioma of carotid body, 5; tumor de cuerpo carotideo, 1; hemangiopericytoma, 1.

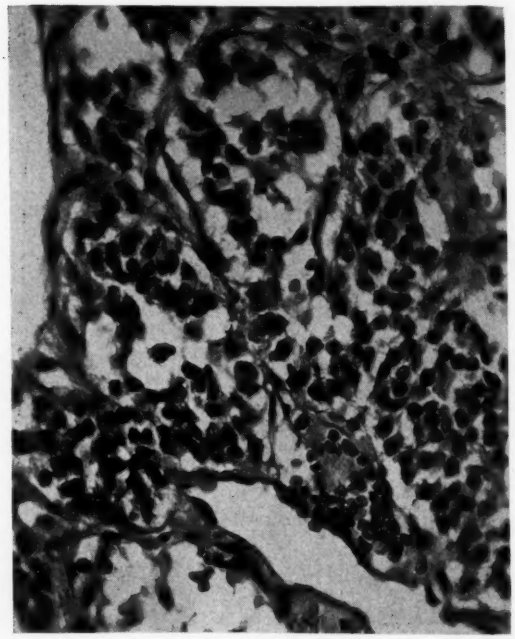

Fig. 1. Case 1. Carotid body tumor. Vessels are surrounded by clusters of uniform cells usually having a clear cytoplasm.

DR. MEISSNER: We are in fair agreement, I see, except for a slight difference in terminology.

It may seem that the inclusion of a typical carotid body tumor seems hardly worthwhile in a seminar such as this. This one is an easily diagnosed tumor. From the history it might be confused with neurofibroma or other similar slow growing tumors, and from the surgical findings it might be confused with a hemangioma or even an aneurysm because of the tendency to such great vascularity. The microscopic appearance, however, is so typical that the diagnosis is not difficult.

The reason for including a tumor such as this, however, is that these tumors are really not very common and none of us sees very many in any one period of time. Furthermore, the pathologist sometimes receives specimens with either an inaccurate, or, more likely, an inadequate clinical history. If a tumor such as this is just sent down as "biopsy of mass of neck," it is possible that, because of their infrequent occurrence, the pathologist may forget to include carotid body tumors in his differential diagnosis. I have seen this happen several times where pathologists were trying to make a diagnosis of metastatic thyroid carcinoma, for example, when the tumor was quite typically one of carotid body origin. They had been misled by inadequate clinical history. Once the possibility of such a diagnosis is considered, it usually becomes easy.

The pathologist is usually asked by the surgeon whether or not the carotid body tumor is malignant. It is true that an occasional carotid body tumor metastasizes to lymph nodes. Rabson and Elliott[14] last year found 10 such cases reported and added an additional one. We have seen only one such instance in our series. Romansky,[16] a few years ago, reviewed the reported malignant carotid body tumors and found 8 cases in which visceral metastases had occurred. He emphasizes, however, that there are no unique changes in carotid body tumors that distinguish the metastasizing tumors from the nonmetastasizing ones. The size of the cells and the presence of tumor giant cells seem to have no particular significance with regard to malignant potential. By far the worst thing about carotid body tumors is the fact that they arise in an anatomic location that makes resection dangerous and difficult.

It is surprising how similar these tumors look to the normal carotid body which shows the same clusters of cells, again accentuated by the silver stain. The current view regarding carotid bodies is that they are chemoreceptor organs and not part of the chromaffin system at all. Therefore, names suggesting that they are chromaffin tumors are misleading. The term chemodectoma is suitable, but it has been difficult for us to utilize this term for clinical diagnostic purposes because the term carotid body tumor is so entrenched in the minds of clinicians and pathologists alike. I do not care for the term nonchromaffin paraganglioma partly because it is a negative diagnosis, and to me somewhat confusing, and partly because carotid body tumors do not act at all clinically like tumors of the soft tissues which are sometimes called by the same name.

DR. SEVERANCE: Col. Lukeman, do you have any follow-up on this case?

COL. LUKEMAN: Eight months after operation the patient was entirely well and there was no evidence of recurrence of tumor.

Case 2 (Glomus Jugulare Tumor)

Diagnosis.—Glomus jugulare tumor (chemodectoma).
Contributor.—Maj. R. E. Kellenberger, M.C., William Beaumont Army Hospital, El Paso.

History.—A 21 year old colored male had a freely movable, rubbery, nontender mass in the postauricular region anterior to the mastoid process of 3 months' duration. A mass lying deep to the jugular bulb and partially overlying the anteromedial aspect of the mastoid was excised. It was circumscribed but apparently not encapsulated and contained many large vascular spaces. Two years before, a polyp had been removed from the ear on the same side which was followed by deafness in that ear.

DR. MEISSNER: We have some additional information on this case from Maj. Kellenberger. Apparently the patient had two operations in 1955, at least one of which was for removal of a polyp from the ear. Major Kellenberger has lent me a slide of this which is composed of granulation tissue covered on one surface by stratified squamous epithelium which is ulcerated. The granulation tissue appears active, but I found no evidence of tumor in this specimen. No tissue is available from the second operation, and it is not even known what was removed.

The tissue submitted for the seminar, which may or may not be a recurrence, looks quite different from the previous specimen. It is a highly vascular mass with rather large foci of fibrosis. Between the vascular spaces are clusters of cells which are epithelioid in nature, fairly uniform in size, and without mitosis. These cells appear, in most instances at least, to be extravascular and not directly related to the endothelial cells of the vessels. In some foci, the cells grow in small clumps which are circumscribed and which are accentuated by silver preparations.

Fig. 2. Case 2. Glomus jugulare tumor (chemodectoma). The lesion is highly vascular with areas of fibrosis.

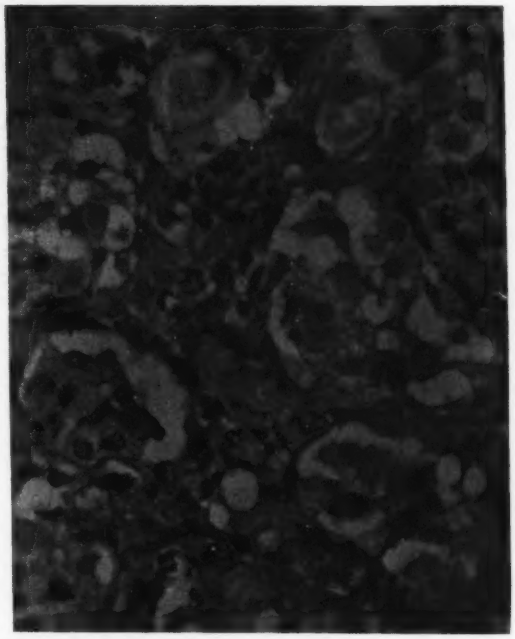

Fig. 3. Case 2. Clusters of epithelial cells similar to Case 1.

We have here, then, a tumor removed from a region of the ear, which is possibly a recurrence, and which shows a few foci of cells similar in nature and arrangement to the cells which we have seen in the typical carotid body tumor. My diagnosis, therefore, is a tumor of the chemodectoma type arising from the glomus jugulare.

This tumor is interesting for several reasons: In the first place, it is not as typical of the chemodectoma group as many. LeCompte,[9] in his fascicle on carotid body tumors, speaks of the "usual" types and of ones deviating from these. This tumor would fit into the latter category. It is easy to see how a tumor such as this, without several sections and without a silver stain, might not be identified correctly. Tumors of the carotid body type may arise in other locations than in the carotid sheath. The most common sites are the region of the aortic arch and the region of the jugular bulb although there are also other locations from which such tumors may arise. Mendelov and Slobodkin[12] have reviewed 7 cases of chemodectoma of the aortic bodies in the mediastinum. Harrison and others,[5] report 9 chemodectomas arising from the glomus intravagale (vagal body tumors) characteristically found just below the

jugular foramen and in continuity with the vagus nerve.

Tumors arising from the glomus jugulare often present initially as a polyp of the ear and it is important, therefore, that all such lesions be examined microscopically. Because of their great vascularity there is, of course, a tendency to ulcerate and bleed and it is not surprising that these tumors may be covered by a layer of granulation tissue and that their true nature may not be disclosed by a superficial biopsy. I think that is probably what happened in this case.

From the morphologic point of view, except for their superficial location and a resulting tendency to become ulcerated or infected, these tumors are similar to the ones arising from the carotid body. However, because of the location, they seem to run a more malignant, or at least a more aggressive, course than those arising from the carotid body, with direct extension and invasion into adjacent bone and even into the base of the brain. As a matter of fact, tumors of the glomus jugulare have rather a poor prognosis. Winship[20] reports 35 cases, in which 12 died either directly or indirectly of their disease. As with the carotid body tumors, cases with metastases have occurred, one of which has been reported by Lattes,[8] to viscera.

It is interesting to note that glomus jugulare tumors show strong familial tendencies. Winship emphasizes also that frequently there are concurrent tumors of the carotid body in the affected patient or in his family.

Submitted Diagnoses.—glomus jugulare tumor (chemodectoma), 18; nonchromaffin paraganglioma of glomus jugulare, 5; recurrent nonchromaffin paraganglioma of glomus tympanicum, 4; carotid-body-like tumor, 3; hemangio-endothelioma, 7; hemangioma (type?, 1; sclerosing, 1, cavernous, 1), 3.

DR. MEISSNER: Chemodectoma seems to be a better name for tumors of this type, and I think "glomus jugulare tumor" with chemodectoma in parentheses would be the way we would diagnose it in our laboratory. The use of new terms sometimes can cause confusion to the detriment of the patient.

I should like to mention one interesting case that we have seen recently. This particular tumor projected into the larynx, and was thought by the otolaryngologist to be a laryngocele. He was surprised to find that he could dissect it free submucosally, and he took out quite a sizable vascular mass without breaking through the mucosa. We think it is an example of a chemodectoma, probably arising from one of the vagal bodies, and projecting into the larynx. One must keep in mind the fact that these tumors can occur in unusual locations.

DR. SEVERANCE: Dr. Kellenberger, do you have any follow-up?

MAJ. KELLENBERGER: Six months after removal this man had deafness, and that was all.

DR. MEISSNER: The deafness, incidentally, according to an ear, nose, and throat specialist with whom I talked about this problem, is more likely due to the evulsion of the stapes in removing the polyp from the ear, than it is to the tumor itself, unless the tumor is very extensive.

Case 3 (Pheochromocytoma)

Diagnosis.—Pheochromocytoma with foci of ganglioneuroma.

Contributor.—Dr. Meissner.

History.—A 50 year old laborer was admitted complaining of progressive abdominal swelling and weakness for 3 years and weight loss for 6 months. There were no other symptoms. Physical examination was negative except for a nontender cystic abdominal mass extending from the pubis to the diaphragm. Blood pressure was 160/92. A 3600 Gm. multicystic retroperitoneal mass filled with red, cloudy fluid and blood clots was surgically excised.

DR. MEISSNER: We have an x-ray on this case which shows a large abdominal tumor with distortion of the ureters, particularly the left.

This was a difficult tumor to diagnose clinically and its exact nature was not even suspected prior to pathologic examination. The mass weighed 3,600 Gm. It was well encapsulated. A flattened adrenal gland lay at one pole. The bulk of the mass was composed of multiloculated cysts filled with blood clot or red, cloudy fluid. The walls of the cystic spaces were smooth, for the most part, but here and there were roughened by adherent blood clot.

Microscopically, the compressed adrenal is adjacent to the capsule and at a few points there seems to be a direct continuity between the adrenal and the mass itself. Otherwise the capsule of the mass appears distinct. Much of it is necrotic, hemorrhagic, and cystic, but at the periphery, particularly, there are identifiable structures. The stroma is collagenous and in some places fibrillar, suggesting the presence of both neurofibrils and Schwann cells. In these foci, particularly, there are large cells with cytoplasmic processes which I think are readily identifiable as ganglion cells. Some of these contain an eccentric collection of brown pigment near the nucleus and one, at least, is in mitosis.

In addition to the ganglion cells, there are other cells of a different type. These occur in clusters, again particularly about the periphery of the tumor but definitely as a component part of the tumor. They are smaller than the ganglion cells on the average. They are polygonal or spherical and the cytoplasm is slightly granular, often containing pigment granules. The pigment here, however, in contrast to that of the ganglion cells, tends to be more diffuse

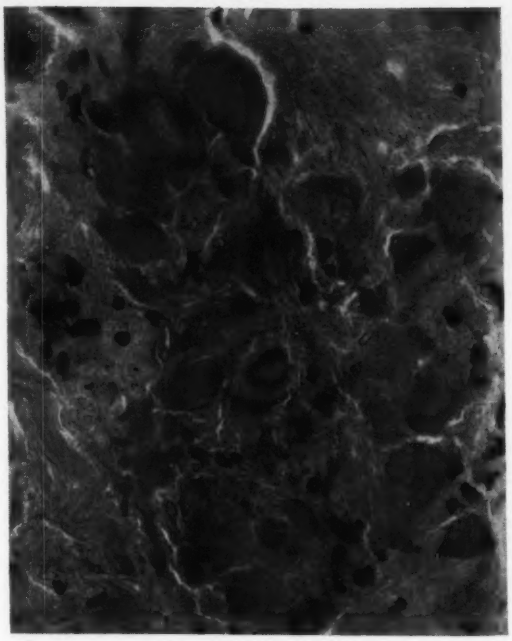

Fig. 4. Case 3. Pheochromocytoma with foci of ganglioneuroma. Nests of large ganglion cells associated with a fibrillar stroma.

and often gives the cells a brown, smudgy appearance. This pigment does not stain for iron, but is accentuated as blue-green pigment with the Giemsa stain after Zenker's fixation. A few of the cells are multinucleated, but I found no mitoses. These cells with the positive chromaffin reaction I think must be identified as pheochromocytes, and the diagnosis, therefore, of this lesion is a pheochromocytoma with foci of ganglioneuroma.

The question often is raised by the clinician whether or not a pheochromocytoma or a ganglioneuroma of the adrenal is benign or malignant. This is admittedly a difficult decision for the pathologist, and I know of no certain way to tell from the morphology alone what the course of an individual tumor will be. Tumors that invade and metastasize, of course, must be considered malignant, but these, as with carotid body tumors, in most instances, do not show a significantly different or consistent morphologic pattern which allows them to be segregated into the malignant group. This particular tumor appeared to be benign from the preoperative and postoperative clinical course. When last heard from, the patient was well and without recurrence after 3 years.

This patient had only slight hypertension preoperatively. Following the pathologic diagnosis of pheochromocytoma, he was observed closely for several days for hypertension, but nothing further was found. The absence of hypertension, paroxysmal or constant, does not, however, rule out the diagnosis of pheochromocytoma since patients with this tumor do not necessarily have hypertension.

The large size of this tumor is interesting. This is by far the largest pheochromocytoma I have ever seen. I suppose the pheochromocytomas that function are recognized clinically and are removed at an early stage, so that development of such a massive tumor is not possible.

While we cannot be certain, it is probable that this tumor did not arise from the adrenal itself. We have been impressed at the relative frequency with which pheochromocytomas arise from accessory or aberrant tissue adjacent to the main gland.

It is interesting to compare these cells with those of the carotid body. It is quite obvious that the pheochromocyte which is a true chromaffin cell is in a different category from the cells of the carotid body.

Submitted Diagnoses.—ganglioneuroma, 27; malignant ganglioneuroma, 3; ganglioneuroma (with nonchromaffin paraganglioma), 2; pheochromocytoma, 3; paraganglioma, 1; hemangiopericytoma, 1; granular cell myoblastoma, 1; mesothelioma (peritoneal), 1; teratoma, malignant, 1.

It is surprising to me that we don't see this combination of cells more often than we do. Since the pheochromocyte and the ganglion cell both arise from the same precursor, there is no reason why one

Fig. 5. Case 3. Groups of pheochromocytes which are smaller than the ganglion cells and contain brown pigment granules distributed throughout the cell.

shouldn't frequently see pheochromocytes and ganglion cells mixed together; but it doesn't happen very often.

DR. LEO LOWBEER, Tulsa: A tumor of this type was shown at the Colorado Springs Seminar in 1957. It originated in the mediastinum and there also was a combination of ganglion cells and pheochromocytes.

DR. SEVERANCE: I would like to ask Dr. Meissner to dissertate a little bit more on the pigment, and various stains that are used on it. I like special stains, occasionally. I once did a Fontana stain on a pheochromocytoma, and it stained nicely positive, giving the reaction that melanin does, although it is not supposed to be that type of pigment.

DR. MEISSNER: We have found that Zenker fixation, without any particular modification, followed by the hematoxylin and eosin stain, often will demonstrate the pigment in pheochromocytes as it did in this case. A Giemsa stain after Zenker fixation, however, is a good easy method to accentuate the pigment, making it greenish-blue. The pigment in a pheochromocytoma is usually not a granular pigment, but imparts a diffuse smudginess to the cells which really is seen better under low power than under high power magnification. This is a different kind of pigment from the pigment in the neurons. We don't know exactly what pigment is; presumably it is a precursor of adrenalin. Often the differential diagnosis in an adrenal tumor is between a pheochromocytoma and an adrenal cortical tumor. Since the cells of these two tumors can look almost identical, only the demonstration of chromaffin pigment may allow the correct diagnosis.

DR. SEVERANCE: If one uses formalin fixation routinely and doesn't think to put portions of one of these tumors into Zenker's solution, what can one do then?

DR. MEISSNER: It isn't as good, but often one can mordant such tissue in a chromate solution and, thus, demonstrate the pigment.

DR. F. P. BORNSTEIN, El Paso: How many of these pheochromocytomas are completely inactive clinically? In the few we have seen it was quite a problem to control the blood pressure at surgery. This seems to be such a bland tumor for a pheochromocytoma.

DR. MEISSNER: I don't know what percentage of pheochromocytomas are nonfunctioning. Like all endocrine tumors, if they are functioning we are more likely to see them as pathologic specimens, because the clinician recognizes them and they are taken out. I think if this tumor had been the size of the usual pheochromocytoma, 5 or 6 cm., it wouldn't have been palpated, and the patient would still have it since it was a nonfunctioning tumor. This explains the large size of the tumor; it wasn't causing clinical symptoms of pheochromocytoma, and, therefore, just continued to grow until it produced symptoms from its size.

Case 4 (Luteinized Granulosa Cell Tumor)

Diagnosis.—Luteinized granulosa cell tumor.

Contributor.—Dr. Robert Hausman, medical examiner for Bexar County, San Antonio.

History.—A 30 year old, obese (5 feet, 3 inches; 190 pounds) Negro cook (gravida IV, para III) had never indicated that she knew herself to be pregnant. One evening while eating candy in bed she lost consciousness and her breathing became heavy and "noisy." She expired in a matter of minutes, and a 3,280 Gm. stillborn fetus was removed from her uterus half an hour later. At autopsy, vomitus was found in the throat and blood-tinged, foamy fluid in the bronchi; the lungs were wet (670 and 500 Gm.). The heart weighed 400 Gm. and had a slightly thickened firm left ventricle. The liver weighed 1,700 Gm. and was somewhat pale. The kidneys (180 and 130 Gm.) were flabby and faintly mottled and striated in the cortex. The adrenal glands were slightly enlarged with hyperplastic cortex. The left ovary (Section A) measured 5 by 3½ by 3½ cm. and was almost completely replaced by light brown, soft nodules, measuring from 0.5 to 3 cm. in diameter. The right ovary (Section B) measured 3.5 cm. in diameter, and 50 per cent of its parenchyma was replaced by similar but smaller nodules.

DR. MEISSNER: Dr. Hausman had been kind enough to furnish me a complete protocol of the autopsy on this case; he has summarized it very well in the abstract. The cause of death was due to toxemia of pregnancy and the findings in the ovaries were apparently more or less incidental discoveries. There were no generalized endocrinologic abnormalities.

From the gross description, we know that both ovaries were nodular and moderately enlarged to about an equal degree. Microscopically, these ovarian nodules appear everywhere identical. They are composed of masses of large, polyhedral, acidophilic cells which have a slightly vacuolated cytoplasm. The nuclei are fairly regular, but mitoses are found without difficulty. The cells form no structural pattern and there is only a scanty stroma. The masses of cells are irregular and at many points appear to be invading ovarian stroma. There are clusters of cells within blood vessels associated with thrombi. The small amount of ovarian tissue remaining in sections shows no abnormality that I could find beyond the presence of these masses just described.

There are two questions in the differential diagnosis—the first concerns the type of cell, and the second the type of process. The differential diagnosis of the nature of these cells lies among three possibilities: first, they are cells of adrenal-cortical rest; second, they are hilus cells; or third, they are lutein cells. These cell types may look quite similar and it may be impossible to differentiate between them.

I have found no Reinke crystalloids, which are supposedly pathognomonic of hilus cells.

The second question concerns the process. The proliferation of the cells is quite active with mitoses relatively frequent. Furthermore, the cells grow within vascular spaces and appear to be invading ovarian stroma. I believe, therefore, that the process is a bilateral tumor which is malignant.

On this assumption, that we are dealing with bilateral malignant tumor, the exact nature of the lesion becomes a little easier. I believe we can rule out adrenal rest tumor and hilus cell tumor for several reasons. In the first place, these tumors are rare; the bilateral involvement would be unusual; and, more important, there is no evidence of masculinization. Without the latter point, I think one cannot make the diagnosis of either of these tumors. This, then, leaves a luteoma as the most likely possibility, and my diagnosis is a malignant luteoma.

If we consider these as lutein cells and the tumor as a luteoma, we probably should speculate as to the origin of the lutein cells. Luteinization ordinarily occurs either in granulosa cells or in cells of the theca interna, and, therefore, when we speak of a luteoma, we are actually speaking of a luteinizing granulosa cell tumor or a luteinizing thecoma. I know of no way to distinguish between these two speculations except that these cells are not as spindle-shaped as usually seen in thecoma. An interesting conjecture, however, is that perhaps this tumor represents a bilateral granulosa cell tumor, which, during pregnancy, became completely luteinized. It is commonly accepted that some granulosa cell tumors do not function, at least from the clinical point of view, so the apparent absence of function of this tumor is in keeping with such a conjecture. The only possible evidence of hormonal imbalance that we have in this case is the obesity. The slight enlargement of the adrenal glands is difficult to assess and could well be explained by the pregnancy.

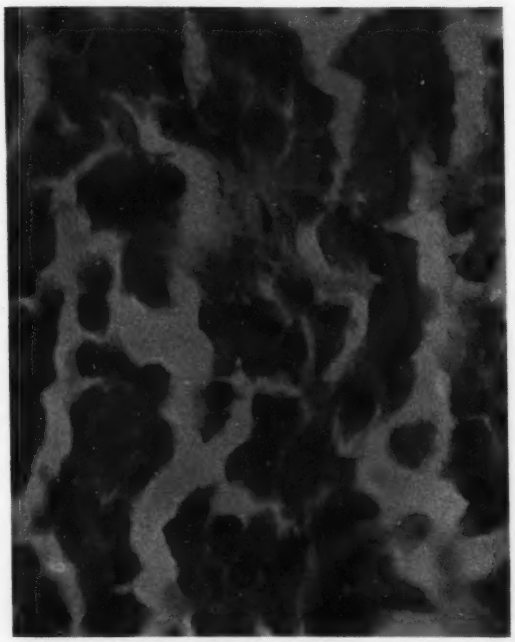

Fig. 6. Case 4. Luteinized granulosa cell tumor. Large polyhedral cells showing occasional mitoses.

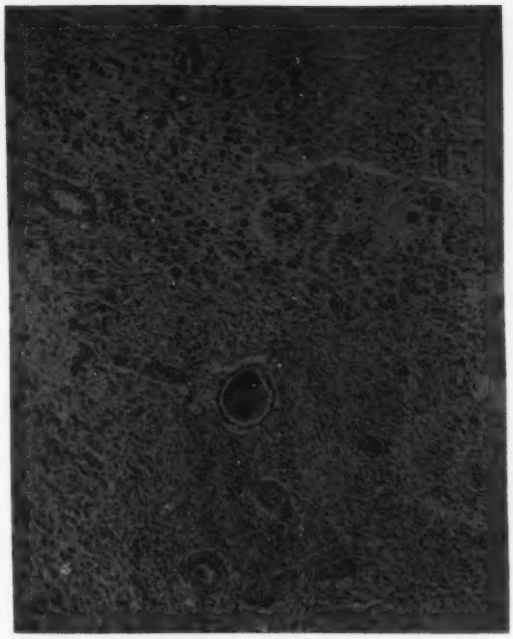

Fig. 7. Case 4. Ovarian stroma is being invaded.

This is another example of how difficult it may be for the pathologist to put a proper name to an endocrine tumor when there is no evidence of hyperfunction. We are all well aware of the fact that it is difficult and often impossible pathologically to estimate from the microscopic appearance whether any given endocrine tumor has produced excessive hormone or not.

Submitted Diagnoses.—adrenal rest tumor, 11; luteoma, 7; adrenal cortical carcinoma, 6; luteinized granulosa cell tumor, 5; granulosa cell tumor, 3; metastatic adrenal medullary tumor, 1; Krukenberg tumor, 1; androblastoma, 1; malignant tumor, type undetermined, 1.

I think an interesting conjecture is the possibility that this woman had a bilateral granulosa cell tumor, perhaps before her pregnancy started, and it was, at least clinically, nonfunctioning. If it had been functioning very much I don't think she could have be-

come pregnant or carried her pregnancy. Then during the course of the pregnancy the bilateral granulosa cell tumor became luteinized. This seemed like an intriguing idea to me, and I tried to find reference to solid ovarian tumors occurring in pregnancy to see if I could find any mention of what happens to a granulosa cell tumor during pregnancy. Solid ovarian tumors as a complication of pregnancy are uncommon, and there are not many reported. Dougherty and Lund[2] in 1950 collected 30 reported cases over a 15 year period and added 6 more cases of their own. There were 3 granulosa cell tumors; but, unfortunately, these were all without microscopic description, so I don't know whether they were luteinized or not.

DR. HAUSMAN: This is the second case that I have seen. The surgeon did a Caesarean section on a woman, found these tumor masses in the ovaries, and decided to do a hysterectomy with bilateral oophorectomy. The woman lived after the operation, and as far as I know was all right. The ovaries were enlarged and had these brown tumors in them. That time I made the diagnosis of luteinized granulosa cell tumor.

DR. MEISSNER: Were there any other endocrinologic abnormalities?

DR. HAUSMAN: There were none, and I particularly looked for them.

DR. J. L. GOFORTH, Dallas: This case intrigued me very much. I had thought that it was a granulosa cell neoplasm showing the luteinizing effects of pregnancy. The problem that worried me was whether it was benign or malignant. I leaned toward the idea that it was malignant, but kept asking myself what would happen if the pregnancy were terminated and the neoplasm were removed. I am not at all sure that it actually is a malignant process.

DR. MEISSNER: I agree with that. I don't know how we can prove whether this is malignant or not. It is like a lot of tumors of endocrine glands—it is difficult to say unless we have a follow-up, whether they are benign or malignant. Our criteria for the diagnosis of malignant change in endocrine glands is more difficult than in many other tissues of the body. This tumor has a lot of mitoses; it looks like it is extending into stroma. But, on the other hand, if this is a tumor of thecal cells or granulosa cells, why shouldn't it extend into its own stroma? It is extending into its own family, really. It is in blood vessels, but again I don't think that necessarily makes it a malignant tumor.

DR. LEO LOWBEER, Tulsa: What did this woman die of?

DR. HAUSMAN: The diagnosis of toxemia of pregnancy was a diagnosis of convenience. I have not the slightest idea what she died of.

Case 5 (Thyroid Hyperplasia)

Diagnosis.—Thyroid hyperplasia (nontoxic).

Contributor.—Lt. Col. James L. Hansen, M.C., Brooke Army Hospital, Fort Sam Houston.

History.—A 22 year old white female had an enlarged thyroid since age 12 with symptoms of hyperthyroidism and hypothyroidism, but at time of surgery seemed euthyroid. Right lobe weighed 30 Gm., left lobe 18 Gm. Cut surface was slightly nodular, moderately firm, and grayish.

DR. MEISSNER: The history in this case is an interesting one of enlargement of the thyroid, with apparently alternating episodes of hyper- and hypothyroidism, for perhaps 10 years. It is significant that at the time of surgical excision the patient was euthyroid. We are told that the gland was moderately enlarged, the two lobes weighing together 48 Gm. We have usually considered that 30 Gm. is the average weight for a normal thyroid gland, although it is true that there are great variations in the weight of the apparently normal gland with the extremes being perhaps as low as 10 Gm. and as high as 50 or 60 Gm.

Microscopically there are two findings of note; one is a diffuse hyperplasia of the epithelium which is present to about the same degree in all parts of every slide. The second feature is the presence of a lymphocytic infiltration which tends to be nodular and often associated with secondary lymphatic nodules. The capsule of the gland is distinct. There is moderate vascularity.

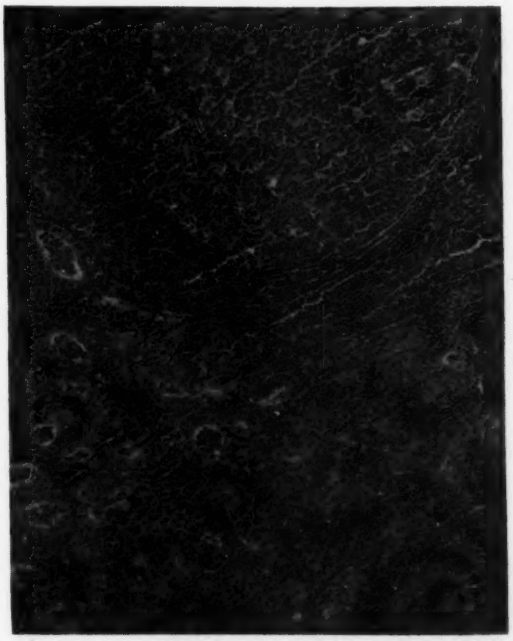

Fig. 8. Case 5. Thyroid hyperplasia, nontoxic. The epithelium is hyperplastic and there is lymphocytic infiltration.

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The differential diagnosis here is between thyroiditis, carcinoma, and diffuse hyperplasia. The diagnosis of thyroiditis does not seem to be a sound one. This degree and this type of lymphocytic infiltration is seen frequently in a gland with hyperplasia of the primary or Graves' disease type. It is true that if the hyperplasia disappears, the lymphocytic infiltration may remain behind, and in such an event the diagnosis of chronic thyroiditis may appear more reasonable. However, in the presence of diffuse hyperplasia, it seems safest to conclude that the lymphocytic infiltration is an accompaniment so commonly seen rather than a primary process. Carcinoma can be excluded by the diffuseness of the process. Presumably, we have representative portions of both lobes and it would be unusual for a carcinoma to be as diffuse throughout both lobes as is seen here. Furthermore, there is no evidence of capsular or vascular invasion. The best diagnosis, therefore, would seem to be a diffuse hyperplasia of the thyroid. I should like to call this primary hyperplasia of the thyroid.

In our laboratory, we are in the habit of distinguishing two types of thyroid hyperplasia. One we call primary hyperplasia and is the type commonly seen associated with the signs and symptoms of exophthalmic goiter. The other we have called secondary hyperplasia, which is the type that occurs in long-standing adenomatous goiter. For clinical reasons it seems important to distinguish between these two types, if possible, because the latter is rarely associated with the exophthalmus, lymphocytic infiltration, or severe clinical toxicity so common in primary hyperplasia. It is a low-grade, smoldering type of hyperthyroidism, difficult to control with antithyroid drugs and the patients are often thyrocardiacs. It is quite a different process from the primary hyperplasia of Graves' disease.

It happens at times that a diffuse hyperplasia of the thyroid occurs without evidence of clinical hyperthyroidism or, as the clinicians say, without toxicity. In order to understand what produces hyperplasia of the thyroid it is interesting to review the physiology of thyroxin secretion. The pituitary under the influence of the hypothalamus produces thyroid-stimulating hormone which stimulates the thyroid gland to hyperplasia and to increased production of thyroxin. The thyroxin, as it leaves the thyroid gland, goes partly to the tissues and partly to the pituitary and hypothalamus where it acts as an inhibitor of TSH secretion. Thus, TSH and thyroxin are normally in more or less of a balance. In the hyperplasia of Graves' disease there is an unexplained hyperplasia of the thyroid and an excessive secretion of thyroxin, but the exact point in the cycle where the difficulty arises is poorly understood. There are cases, however, where the thyroid may be unable to put out either sufficient or functionally useful thyroxin, in which case the pituitary would not be depressed and the excessive TSH, therefore, stimulates the thyroid to greater and greater hyperplastic activity in the presence of clinical euthyroidism or even hypothyroidism. Such a state may be brought about by several mechanisms. For example, there are so-called goitrogenic substances, such as thiocyanate, which inhibits the iodine trapping mechanisms of the thyroid or thiouracil, which prevents the intraglandular formation of thyroxin. There are other goitrogenic substances, the mechanisms of which are poorly understood. Still, in addition, there may be an inborn fault in thyroid metabolism, for example, with one of the intrathyroid enzyme systems, so that a usable thyroxin is not adequately formed. This is, perhaps, the explanation of some cases of congenital goiter.

In any event, the pathologist occasionally is confronted with a case where there is no evidence of clinical hyperthyroidism, but where the thyroid gland is nevertheless diffusely hyperplastic. For want of a better name we have been in the habit of calling such a gland pathologically primary hyperplasia, nontoxic.

Two examples will serve to illustrate this type of hyperplasia. The first case is one of a goiter in a 3½ month old child where there is a diffuse hyperplasia of such an extreme degree that the baby died from respiratory obstruction. The cause for the hyperplasia in this instance is unknown, but the mother did receive desiccated thyroid during the last trimester of pregnancy, and the child received iron and cobalt the first few months of life. The second example is from an adult with recurrent enlargement of the thyroid and without clinical toxicity. Here again there is a diffuse hyperplasia which is quite similar to the hyperplasia found in typical Graves' disease. In this case, again, there was no obvious reason for the thyroid enlargement.

This nontoxic hyperplasia of the thyroid is uncommon, but is important for the pathologist to remember. In the absence of clinical hyperthyroidism it is sometimes tempting to call such a process carcinoma. In the case we have just discussed, I mistakenly made a diagnosis of carcinoma on frozen section only to find on our permanent sections that the gland was diffusely involved with nothing but a hyperplastic process. It is possible that some of the thyroid cancers reported in infants are also instances of an overdiagnosis of diffuse hyperplasia.

Submitted diagnoses.—chronic thyroiditis, Hashimoto, 20; chronic thyroiditis nonspecific, 5; Riedel's struma, 1; hyperplasia with involution and lymphoid infiltration, 3; atypical thyroid hyperplasia, 2; Hashimoto's disease associated with fetal adenoma of thyroid, 1; toxic goiter (Gulf Coast?), 1; follicular Hurthle cell adenoma, 1; adenocarcinoma, 3; follicular adenocarcinoma, 2; papillary adenocarcinoma, 1.

DR. MEISSNER: Hyperplasia with involution and lymphoid infiltration would be similar to my diag-

nosis; atypical hyperplasia would also fit. I won't argue with anybody who calls it Gulf Coast goiter, because you all know more about that than I do. I didn't see anything to suggest an adenoma here, and I think we already have ruled out carcinoma.

I am impressed by the fact that 20 people thought this was Hashimoto's disease. Hashimoto's disease, it seems to me, is a diagnosis that cannot be made from lymphocytic infiltration. It must be made from the epithelium. Lymphocytic infiltration occurs in many conditions, and if we call a process Hashimoto's disease only because there are lymphocytes, we can make the diagnosis of Hashimoto's disease on about half the thyroids we see. The important thing in Hashimoto's disease is the nature of the epithelium, which is an acidophilic, sick-looking epithelium. It doesn't look hyperplastic; it looks as though it isn't functioning at all. Of course many patients with Hashimoto's disease have hypothyroidism. As you know, there is a recent theory that Hashimoto's disease is perhaps caused by an autoantibody against the person's own thyroglobulin, with a resultant reaction within the gland. This gland is much too active, it seems to me, to be a Hashimoto's disease, and I would not make the diagnosis from the presence of just lymphocytic infiltration. In patients with Graves' disease the lymphocytic infiltration is often much more diffuse and much more extreme even than in this case here, but the important thing is that the epithelium is hyperplastic, not acidophilic and hypoplastic.

Chronic thyroiditis is also a reasonable diagnosis, in the differential at least, because of the lymphocytes. If we took the hyperplasia away, I think we could call this a chronic thyroiditis. Some people have suggested that when one has Graves' disease with a lymphocytic infiltration, and then the hyperplasia disappears, one has then a Hashimoto's disease. I don't think, however, that Hashimoto's disease arises from a previous bout of Graves' disease. It seems to be an entirely different process. A few years ago we reviewed over 100 cases which we thought met the criteria of Hashimoto's disease as Hashimoto described it originally, and we were impressed by the fact that hardly any of these patients had even a suggestive history of previous hyperthyroidism. This theory has been suggested many times, but I don't think it is a very good one.

DR. LEO LOWBEER, Tulsa: Dr. Meissner, I wonder how you got the preparations for demonstrating the typical changes in hyperthyroidism, because we never see any such glands prior to treatment, and, therefore, don't know what is due to the disease and what is due to the treatment. Another case you showed is an example of the papillary type of hyperplasia in nodular goiters that we see quite frequently in entirely non-nodular goiters, all of which have been treated with iodine prior to the operation.

DR. MEISSNER: I suppose it is true that we must incriminate iodine as a possible goitrogenic agent. Certainly, we know that iodine given in large doses sometimes produces goiter, for example, in patients who take iodine for bronchial asthma. It also is true that we rarely see a patient with hyperthyroidism who has not been treated. Now that thiouracil also is being used, the subject is even further complicated for pathologists. However, we can rely on the descriptions of the hyperplastic gland before iodine was used, and the glands that were seen prior to the introduction of thiouracil.

DR. J. J. ANDUJAR, Fort Worth: Since we don't have another case of Hashimoto's disease in the group today, I am wondering, before we leave the subject, whether you would comment on the significance of the amount of lymphoid stroma necessary for this diagnosis.

DR. MEISSNER: The presence of numerous lymphocytes and secondary lymphatic nodules should always make one think of the possibility, but beyond that I don't think the degree of lymphocytic infiltration is of any value in the diagnosis. The diagnosis must be made from the epithelium, and that is even more important now if the idea of autoantibodies is a correct one.

DR. DANIEL L. ROSENSTEIN, San Antonio: You said in discussing the epithelium in Hashimoto's disease that it is oxyphilic—I quote you, "kind of sick." This is a little uncertain to me yet. I gather that a lot of other people felt a little uncertain about this particular case. I feel that the epithelium here is oxyphilic; on the other hand, as you point out, it is hyperplastic. Would you be a little more detailed in describing the changes?

DR. MEISSNER: The typical epithelium in Hashimoto's disease is a low cuboidal oxyphilic epithelium. The cells look like the Hurthle cells very much, and perhaps the Hurthle cell is a thyroid epithelial cell which has retrogressed, or is not as capable of function as the normal thyroid cell. The oxyphilic cell in the thyroid compares with the oxyphilic cell in the parathyroid—a cell that is not capable of much function. There is practically no colloid in the typical case of Hashimoto's disease, but it isn't because there is no room for colloid. There is no room for colloid here in this case because the cells are all so hyperplastic that the follicles are small and tight. In Hashimoto's disease there are follicular spaces that can be found, but there is little or no colloid within the spaces. The diagnosis of Hashimoto's disease I think has been made far too often by us pathologists and as a result the diagnosis has been diluted down until it really doesn't mean much. If we just make the diagnosis of Hashimoto's disease from finding lymphocytes we get to the problem that was raised a

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few minutes ago—how many lymphocytes do you need? Seventy per cent, or fifty per cent, or ten per cent? If one goes over a series of specimens and compares the clinical history and the follow-up, the gross findings, and the pathologic microscopic findings together, one can arrive at a fairly concrete clinicopathologic entity which is Hashimoto's disease. But if one diagnoses Hashimoto's disease just because of lymphocytes, the correlation with clinical findings is poor.

DR. SEVERANCE: I notice that Dr. Andujar was perturbed because we didn't include a case which Dr. Meissner would diagnose as Hashimoto's disease. I note that 20 other people around here did think that this case was Hashimoto's, and we've had enough discussion so we probably don't need one in this seminar. This probably goes back to the fact that a good many of us here, including myself, were led to believe, or were taught somewhere that a lot of lymphocytes did give you a good picture of Hashimoto's, and maybe we weren't taught to stress the other feature and the clinical picture. We have trouble getting the clinical information; it is usually not readily available unless you get the chart several weeks or maybe a month after the patient has gone from the hospital. Maybe that is different in Boston.

DR. MEISSNER: Not very much.

Case 6 (Chronic Thyroiditis)

Diagnosis.—Chronic thyroiditis, nonspecific.

Contributors.—Drs. D. A. Todd, A. M. Richmond, and Sylvia Johns, Nix Hospital Clinical Laboratory, San Antonio.

History.—This white female, 41 years of age, first noticed enlargement of the neck in February, 1957. Physical examination revealed a large adenomatous growth in the right lobe. Pulse was 82, blood pressure 140/80. Radioactive iodine uptake study with 50 microcurie tracer dose showed 17 per cent concentration over the thyroid in 24 hours with practically no uptake in the right lobe. A total thyroidectomy was performed in March, 1957. The right lobe was found to be firm, pale red, and diffusely lobular with no discrete nodules. The left lobe, on section, presented a red background with firm, gray-white, indurated areas.

DR. MEISSNER: The history does not help a great deal in the diagnosis of this interesting case. We are told that the 41 year old woman had enlargement of the thyroid, especially the right lobe, of rather short duration, and that the radioiodine uptake in the thyroid was rather poor. The two lobes removed at operation are nondescript and more or less similar in appearance.

Microscopically the tissue shows four features. In the first place there is a considerable fibrosis of the thyroid; secondly, there is an inflammatory cell infiltrate composed of lymphocytes and plasma cells; thirdly, many of the thyroid follicles are distorted and devoid of colloid; and, fourthly, some of the epithelial cells also are distorted and frequently contain large or multiple nuclei. All of these changes are present to slightly varying degrees throughout the tissue.

The differential diagnosis here lies between chronic thyroiditis and carcinoma. I think we can all agree that there is at least a chronic thyroiditis present; the problem that then arises is whether or not there is a coexistent carcinoma. My diagnosis is thyroiditis without carcinoma. In the first place, the stromal fibrosis does not seem to me to be either an inherent part of a tumor or to be invaded by tumor; rather it suggests that the epithelial cells are being distorted by the fibrosing process. Secondly, the inflammatory infiltration has no constant relationship with the thyroid epithelium, and, therefore, suggests a primary process rather than one secondary to tumor. Thirdly, the distorted irregular follicles suggest that the distortion has been caused by stromal changes rather than atypical growth. Fourthly, the cells with atypical nuclei, while sometimes seen in thyroid tumors, also are seen in adenomatous goiter and various types of chronic thyroiditis, often being referred to as cells atypical because of anoxia; and, lastly, I find no evidence of invasion of stroma or vessels by epithelial cells. This last point is of most significance since it is possible for both thyroid follicles and cells to be atypical under many benign conditions. In such an event, the diagnosis of cancer can only be made with certainty by finding definite vascular or stromal invasion.

I had the opportunity, if not the advantage, of seeing this case a year or so ago at which time Dr. Todd sent it to me for my opinion. I was happy to find that my diagnosis this time of chronic thyroiditis was similar to the opinion that I expressed when I saw the slides last year. If my diagnosis is wrong, at least I can feel that I have been consistent. I hope that Dr. Todd will have some follow-up information on this case which will prove to be of interest.

A differential diagnosis between chronic thyroiditis and carcinoma may be extremely difficult. Carcinoma is accompanied frequently by chronic thyroiditis, and sometimes it appears that cancer arises in a gland previously involved with chronic thyroiditis. We have adopted a general rule which has helped us in most instances, particularly on frozen section diagnosis, that if the differential diagnosis lies between chronic thyroiditis and carcinoma, then one must make the diagnosis of carcinoma with extreme caution and only with obvious criteria. This rule has rarely failed us in practice.

The cause of such a thyroiditis as seen in this case is difficult to conjecture. As in many inflammatory processes, once the disease has been present for a time the causative factors are often no longer distinguishable.

Any discussion of thyroiditis is difficult, and a good classification of thyroiditis does not exist. There

are several reasons for this. In the first place there are certain diseases which have been included under the heading of thyroiditis which are probably not true inflammations. In such a category is struma lymphomatosa or Hashimoto's disease. Perhaps in the same category comes the lymphocytic goiter which is somewhat similar to Hashimoto's disease, but without the extensive epithelial changes usually found in the latter. A further complicating factor in the classification of thyroiditis is that an etiologic classification is difficult. Even in the types of thyroiditis which we can recognize reasonably well pathologically, such as granulomatous thyroiditis, we still do not know the etiologic agent.

Still another complication is due to the fact that we often see the end results of injury to the gland and for want of a better name label these as nonspecific chronic thyroiditis, even though we have no idea as to what the injurious agent might have been.

I think it is in the latter group that the case today falls. We really have little to go on in this type of thyroiditis except fibrosis and inflammatory cell infiltration. These certainly are nonspecific entities and may be the end result of any number of causes, all unrecognizable, at least in the stage in which we see a gland such as this. To the more common entities included under chronic thyroiditis, the pathologist often is able to give a name, but we all appreciate

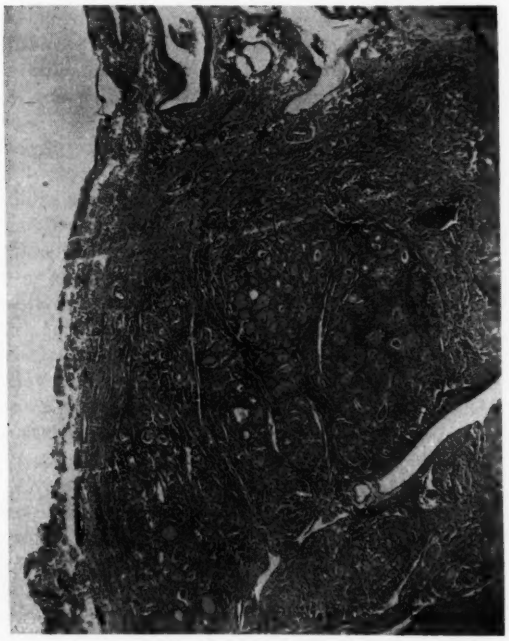

Fig. 9. Case 6. Chronic thyroiditis, nonspecific. There is considerable fibrosis.

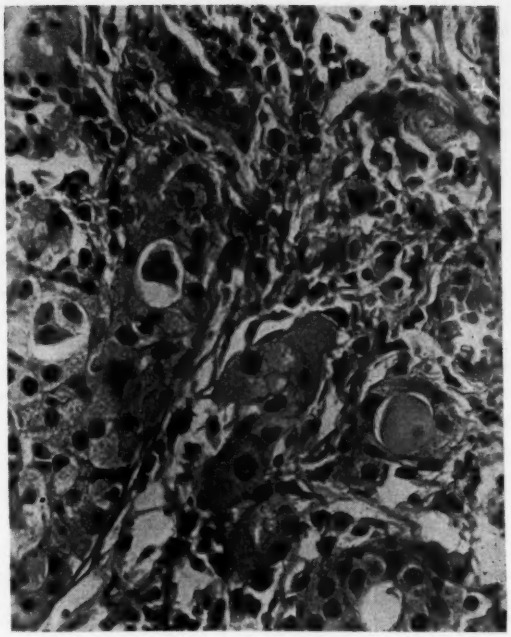

Fig. 10. Case 6. There is an inflammatory round cell infiltrate and the follicles as well as the epithelial cells are distorted.

the fact that there are many cases where no specific name can be applied, and for these the term nonspecific chronic thyroiditis must be used.

Submitted Diagnoses.—chronic thyroiditis, 11; thyroiditis with Hurthle cell, 8; granulomatous thyroiditis, 3; chronic thyroiditis, Hashimoto type, 3; Riedel's struma, 3; nodular hyperplasia, 3; subacute thyroiditis, 2; adenocarcinoma, 3; follicular adenocarcinoma, 3; papillary adenocarcinoma, 1.

DR. MEISSNER: We have discussed some of these differential diagnoses already: chronic thyroiditis—nonspecific, I suppose that implies; thyroiditis with Hurthle cells—I'd lump these first two together under one heading. Granulomatous thyroiditis is a different process from this; it is the subacute thyroiditis where there is a giant cell reaction to the breakdown of the colloid to give a real granulomatous change.

Again we have Hashimoto's disease suggested, but this case does not fulfill the necessary criteria. Riedel's struma is an extremely rare disease in the thyroid. It does not occur more than once in every several thousand thyroid specimens if one uses the diagnosis as Riedel used it when he described his original cases—a diffuse fibrosis of the thyroid with extension of the fibrous tissue out into the adjacent structures of the neck. If one uses the diagnosis of Riedel's struma for just a little fibrosis of the thyroid, you can use it on about half of the thyroids; and if you use lymphocytes to diagnose Hashimoto's disease, you

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could use that on the other half. I wondered in this case if perhaps the patient had not had a previous episode of hyperthyroidism with a resultant lymphocytic infiltration and a little fibrosis that often occurs with involution, so that this represents the end process. This is one way that the nonspecific type of thyroiditis that we see here could be explained. I wish I knew a good classification of thyroiditis. I certainly do not have one, and I do not know of any very good one that anybody has.

DR. RICHMOND: So far there is no evidence of carcinoma in this case. I had the privilege of seeing a frozen section on this particular case a little over a year ago. The tissue I received from the surgeon was a small, encapsulated nodule that he said was discretely separated from the thyroid and lay along the inferior border. I told him at that time if he were sure it was separate, it was likely carcinoma. Later I had to eat my words.

DR. MEISSNER: The fact that there was an accessory piece of thyroid tissue outside the main gland I think helps support the suggestion that there was a previous hyperplasia at one time. Frequently in a Graves' disease type of hyperplasia, prolongations of the thyroid epithelium push out into the adjacent structures, even into the muscle, and may remain behind after the hyperplasia is over. It is tempting to call these residual foci carcinoma, especially if there is no history of previous hyperthyroidism.

DR. GEORGE MANI, San Antonio: In Case 5 the differential was carcinoma versus hyperplasia versus thyroiditis, and in this case it is again carcinoma versus thyroiditis in the differential. In each case we had the benefit of seeing the entire gland. How sure can one be of the diagnosis when you see only a portion of the gland submitted by the surgeon? Also when you receive the entire gland, how many sections do you take before you feel somewhat certain of your diagnosis?

DR. MEISSNER: That is a good point. It is worthy of emphasis that if one just receives a biopsy from a gland like this and sees this sort of epithelium with its atypical cells and the surgeon says it is from outside the thyroid gland, quite a few of us might be taken in. When you have the whole gland for examination, it is much easier. The number of sections that one should take depends on the gross appearance of the gland. If the process appears diffuse throughout both lobes, two, three, or four blocks should be ample, but if there is something which looks grossly suspicious of tumor, then one might want 10 or 15 blocks; there is no hard and fast rule.

DR. SEVERANCE: In order to save myself and others a lot of worries and arguments, would it be possible for you in a brief fashion to run down the various points of differential microscopic diagnosis between these forms of thyroiditis to consolidate it in our minds?

DR. MEISSNER: There are two or three specific forms of thyroiditis that are relatively easy for the pathologist to diagnose. One is acute, suppurative thyroiditis, which should give no diagnostic trouble but which pathologists seldom see because it is not treated by excision. The types of thyroiditis that we see are the ones that for the most part are confused clinically with carcinoma.

The type called granulomatous thyroiditis, or subacute, is the one that has a history typically of 6 weeks to 2 months' duration. This type of thyroiditis produces a breakdown of colloid with a resultant foreign-body reaction to the broken-down colloid. While this process seems to be due to an infection, cultures are sterile. The typical change is the formation of abscesses within follicles with the subsequent breakdown of colloid, which seems to act as a foreign material. The foreign body giant cells cause a resemblance to tuberculosis—pseudotuberculosis is a synonym. This is a rather specific type of thyroiditis, clinically and pathologically, and can be diagnosed as such in most instances. The clinician also can make the diagnosis if he thinks of it. Characteristically these glands have no iodine uptake and are associated with a high sedimentation rate. We have been surprised at the low incidence of preoperative diagnosis of chronic thyroiditis. It runs about 30 per cent.

Another type of thyroiditis is Hashimoto's disease, if one wants to call this a thyroiditis. As we have already discussed, there is a good question whether this should be included as a true thyroiditis, but there is no other convenient place to put it in the thyroid classification so I suppose we can leave it there. It almost always occurs in females and most commonly around the menopausal age, which suggests to me some endocrine imbalance as a factor in its development.

There is another type of thyroiditis rather similar to Hashimoto's disease which does not have a good name as yet; it shows a lymphocytic infiltration similar to that seen in Hashimoto's disease, in a variable degree, but the epithelium does not look as acidophilic. It is seen in younger people as well as in older people and represents either an earlier stage of Hashimoto's disease or probably a slightly different process. This is the lymphocytic goiter without the typical epithelial changes of Hashimoto's disease. Tuberculosis, syphilis, and specific inflammations of the thyroid are rare.

When we consider the types of thyroiditis about which we feel reasonably secure diagnostically, there is the granulomatous or subacute thyroiditis, Hashimoto's disease, lymphocytic goiter, and Riedel's struma. Most of the cases of chronic thyroiditis that we see do not fall into any of these categories. They are just a nonspecific chronic thyroiditis, like the case we saw here, and we do not know the cause. I should like to emphasize again that fibrosis in itself

does not mean Riedel's struma and lymphocytes by themselves do not mean Hashimoto's disease.

DR. D. L. GALINDO, San Antonio: Do you routinely do frozen sections on these questionable thyroid lesions?

DR. MEISSNER: The question of the value of frozen section in thyroid is one that always comes up among the surgeons. We think that it is worth doing although admittedly there are times when, if you are honest, you must say, "I do not know." On a frozen section if I am sure there is definite chronic thyroiditis, I am reticent in making the additional diagnosis of carcinoma.

DR. J. H. CHILDERS, Galveston: Several years ago there were a number of surgeons who advocated partial thyroidectomies and subtotal thyroidectomies in the treatment of patients with moderately severe hyperthyroidism and a number of other conditions. Have you had an opportunity to review sequential biopsies in this manner?

DR. MEISSNER: We have had a chance to see some of these because, of course, with any therapy for Graves' disease, whether it is radioiodine therapy or surgery, there is a certain percentage of cases that will have a recurrence or two or three recurrences. These glands do not progress to Hashimoto's disease. What they do progress to is a fibrotic gland—not an extreme fibrosis but an amount of fibrosis about equivalent to that in the case here. This seems to be accentuated with every recurrence of the hyperthyroidism along with some lymphocytic infiltration, so the final result is a microscopic appearance that looks like either a nonspecific chronic thyroiditis or a mild adenomatous goiter.

Case 7 (Follicular Carcinoma)

Diagnosis.—Follicular carcinoma.

Contributor.—Capt. D. R. Snyder, M.C., Brooke Army Hospital, Fort Sam Houston.

History.—A 50 year old white male was found on physical examination to have asymptomatic enlargement of thyroid. The right lobe contained a 5 by 3 by 2 cm., apparently encapsulated, firm mass, the cut surface of which was indistinctly nodular and composed for the most part of pink, fleshy tissue. Two follicular adenomas also were found in the opposite lobe.

DR. MEISSNER: We are told that grossly this is an encapsulated nodule. Microscopically, the mass is composed of a collection of thyroid cells which tend to form small follicles usually without colloid. The cells individually are usually a little larger than the normal thyroid cells, somewhat acidophilic, and some of them have nuclei considerably larger than the normal. An occasional cell shows a mitosis. At the periphery of the mass, there are several prolongations

Dr. William A. Meissner is a pathologist at New England Deaconess Hospital, Boston.

of these cells into the adjacent fibrous capsule and even into the adjacent thyroid gland. These prolongations seem to be direct invasions rather than just inclusions since the cells in these foci appear to be actively proliferating.

In addition, there are foci where the tumor is growing rather intimately with blood vessels and lymphatics and, while I strongly suspect that both are invaded by tumor, I could not find a field good enough for a convincing photomicrograph. However, we can all agree that this nodule is a tumor and that it is at least invading through its capsule and is therefore malignant.

My diagnosis is follicular carcinoma.

Submitted diagnoses.—follicular adenocarcinoma, 12; follicular and papillary adenocarcinoma, 3; Hurthle cell carcinoma, 2; papillary adenocarcinoma, 1; small cell carcinoma, 1; adenocarcinoma (type?), 5; adenoma (type?, 4; embryonal, 5; Hurthle cell, 3; fetal, 1; follicular, 3) 16.

DR. MEISSNER: I think I would need to see a little bit more in the way of papillary foci to call this a mixed papillary and follicular carcinoma. Maybe other people had more of this on their slides and this diagnosis, then, would be in order. Without the capsular invasion demonstrated one would have to leave this as an adenoma, benign rather than malignant.

There are several interesting points for discussion in a lesion such as this. In the first place the nomenclature of these tumors which clinically and grossly seem to be benign adenomas, but which turn out microscopically to have some foci of carcinoma present, presents a difficult problem for the pathologist. They have been called by many different and confusing names; for example, potentially malignant adenoma, adenoma with minimal cancer, adenoma with early cancer, and adenoma with invasion. None of these terms is satisfactory, but from the clinical point of view it seems rather important that these cases be placed in a separate group from well-developed adenocarcinomas. This is because such tumors infrequently metastasize and many consider that radical surgery is unnecessary for treatment of them. It is a similar problem to the one that arises when we find a small focus of cancer in an adenomatous polyp of the rectum—it is not always necessary to do a radical resection for such a lesion. In lieu of a better

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term we have used "adenoma with invasion" as a descriptive one, actually to place the follicular carcinoma in a minimal stage and, thus, assist our surgical colleagues in deciding whether or not radical surgery should be performed. The tumor in this seminar case, however, I believe shows too much activity and particularly too much invasion of the capsule to allow it to be considered in this minimal stage of follicular carcinoma.

It also is always interesting to speculate as to whether these are cancers arising from preexisting adenomas or whether they were cancers from the beginning. We pathologists have been confused in our thinking about this since it has been a common habit of the clinicians to call all nodules of the thyroid adenomas. If such a nodule is removed and cancer is found in it, it unfortunately has been assumed that it was cancer arising in an adenoma. A few years ago McManus and I[11] studied a series of 500 adenomas and were impressed by the rarity of papillary adenoma, in spite of the fact that papillary carcinoma is by far the commonest single type of thyroid cancer. We were impressed further by the fact that the average age at time of removal of adenomas and carcinomas is about the same. These facts lead us to conclude that most of the papillary carcinomas do not go through a benign tumor stage. Furthermore, it is difficult for the pathologist to prove that follicular carcinomas arise from a preexisting follicular adenoma, even though it may be suggested in many cases. The important clinical conclusions would seem to be that a solitary nodule should not be removed primarily because it may become cancer in the future; it should be removed—just as a solitary nodule of the breast—to see whether or not it is cancer already.

Adenomas may be present for a long time and not become cancer.

Dr. Rathmell, a few years ago, sent me a case which is an excellent example of a benign adenoma. It is also an example of how an adenoma may be present for a long time and still not turn into cancer. This nodule was removed from the patient at autopsy, and the patient was 100 years old when she died. Although the tumor had been present since the age of 20, after 80 years it was still quite benign.

One other point of importance concerns the selection of blocks by the pathologist when he examines a grossly circumscribed thyroid nodule. In most instances there is little value in examining the central portion of the nodule; not only are degenerative changes frequent and confusing, but many thyroid cancers, from cellular morphology alone, are indistinguishable from adenomatous goiter or even from relatively normal thyroid. The important thing, therefore, is to examine the periphery of the tumor and to look for invasion in this area. This is where invasion is most readily found, either in the capsule, in the adjacent gland, or in vascular spaces. This is particularly important since many thyroid nodules can be diagnosed as cancer only by finding invasion.

DR. J. L. GOFORTH, Fort Worth: In reference to terminology, most clinicians will refer to any nodule in the thyroid as an adenoma. Do you distinguish between the adenoma which would mean, strictly speaking, a true neoplasm, on the one hand, and the nodular hyperplasia on the other; and if you do, do you think the one is more likely to undergo malignant change than the other?

DR. MEISSNER: We do try to distinguish between the two, although admittedly it is a hard thing to do in many instances. If there are multiple nodules we think that it is probably not a neoplasm, but a part of a nodular goiter, or adenomatous goiter. There is more evidence that carcinoma arises in a true neoplastic adenoma than in a multinodular goiter, and for that reason, and perhaps only for that reason, it is rather important to distinguish between them.

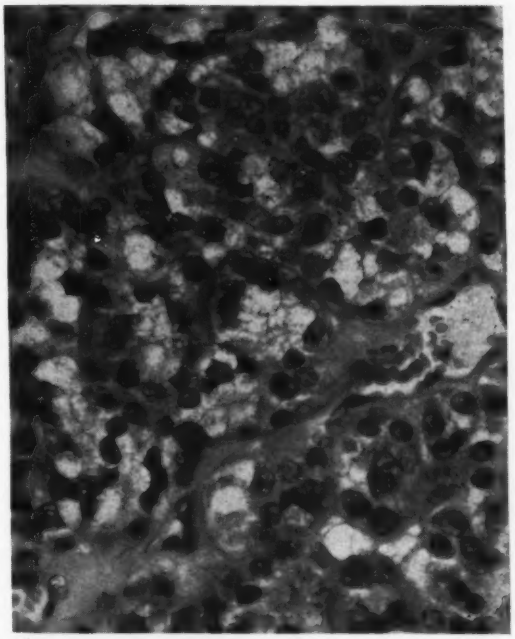

Fig. 11. Case 7. Follicular thyroid carcinoma. Moderately large pleomorphic cells tending to form follicles, usually without colloid.

DR. GEORGE MANI, San Antonio: How often, if ever, do you see follicular carcinomas metastasizing as papillary carcinomas?

DR. MEISSNER: I don't think follicular carcinomas metastasize as papillary carcinomas unless they have a papillary component in them somewhere. We recently reviewed 17 cases of carcinoma of the thyroid with persistent tumor for 10 or more years;[10] in

other words, we compared the original biopsy with tumor removed again after 10 to as long as 26 years. We were impressed by the fact that only one of the 17 tumors changed its type, and this one changed from a follicular carcinoma to a giant cell carcinoma.

DR. LEO LOWBEER, Tulsa: With reference to changing type, we saw recently a carcinoma in which the primary tumor was presumably follicular, with occasional papillary areas, and 3 years later the tumor recurred as a spindle cell carcinoma almost unrecognizable as a carcinoma.

DR. J. B. HUTCHESON, Dallas: We run into trouble on occasion when we use the term adenomatous goiter, because the surgeons think that this is an enlarged gland which contains multiple adenomas. Occasionally we see a goiter of this type which contains what we consider to be actual adenomas, and as a consequence we have begun using the term nodular goiter, and have avoided using the term adenomatous goiter. Don't you think it would lead to a better understanding among surgeons and pathologists if we would eliminate this term adenomatous goiter?

DR. MEISSNER: I certainly agree. I don't like the term adenomatous goiter very well, but nodular goiter isn't any better, because a lot of processes will produce a nodular goiter, including chronic thyroiditis, benign and malignant tumors, and iodine deficiency.

DR. C. P. SCHWINN, Houston: We are always having trouble with the Hurthle cell adenoma versus Hurthle cell carcinoma. I wonder if you have any diagnostic criteria other than the usual to use in this particular situation.

DR. MEISSNER: I don't think there are any. The same criteria hold for the Hurthle cell tumors as for any other type of tumor. As a matter of fact, I am not sure it is necessary to distinguish Hurthle cell tumors as a separate group. They probably can be incorporated with follicular carcinomas and just left like that. It makes it much simpler, and certainly they act about the same as the follicular carcinomas clinically.

Case 8 ("Atypical" Thyroid Carcinoma)

Diagnosis.—"Atypical" thyroid carcinoma.

Contributor.—Col. J. M. Lukeman, M.C., Fourth United States Army Medical Laboratory, Fort Sam Houston.

History.—This 37 year old woman of Caucasian-Indian descent first noticed thyroid enlargement approximately 4½ years ago. The thyroid gland was said to have increased gradually in size, and the patient stated that she noted increasing heat intolerance, nervousness, and some associated weakness. In December, 1956, a diagnosis of toxic goiter was made, and the patient was treated with propylthiouracil, showing marked symptomatic improvement. During therapy the gland enlarged rapidly, then progressively regressed until it was definitely smaller than at the onset of therapy. A nodule, however, persisted in the left lobe. At operation a large, completely encapsulated tumor was found. Physical examination and x-ray survey at that time revealed no evidence of enlarged nodes of the neck or of metastatic tumor. The specimen received in the laboratory measured 7 by 4.5 by 4 cm. and weighed 35 Gm. The cut surface in the fixed state was gray-tan with hemorrhagic areas. Almost the entire lobe consisted of this grayish firm tissue with a thin capsule which revealed in several areas brown, glistening tissue resembling thyroid structures. There were likewise areas that cut with grittiness suggestive of calcification.

DR. MEISSNER: From the history of this case we find that the patient, with a relatively recent history of hyperthyroidism controlled with thiouracil, had a 35 Gm. encapsulated mass removed from one lobe.

At the periphery of the tumor there is a capsule which is well defined and intact in most areas. However, in at least one focus there is extension of the cells of the mass through the capsule and into the adjacent thyroid.

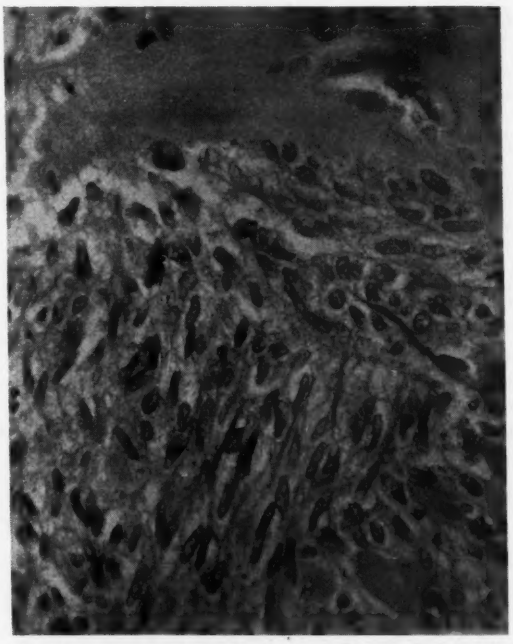

Fig. 12. Case 8. "Atypical" thyroid carcinoma. The major portion of the tumor presents a sarcomatous picture with many foci of pink-staining material which appears to be secretion.

Microscopically the mass is composed of cells which in part appear epithelial, but for the most part are spindle-shaped and suggest a connective tissue derivation rather than epithelial. In only a few areas are the cells lined up in a follicular pattern. There is some nuclear variation, and a rare mitosis is found. Throughout the tumor are foci of pink-staining material which is afibrillar and which ap-

pears to be more a product of abnormal secretion than a true fibrosis. This material is intimately connected with the cells of the nodule.

The problem here is somewhat similar to the problem of the previous case. I think we can conclude that this is an invasive tumor. The problem is one of deciding on the proper name for it.

Benign tumors similar to this encountered in the thyroid gland have been called by Hazard[6] "atypical adenomas." They form no colloid nor do they form even a good follicular arrangement. Moreover, the spindle cell nature may make them appear more like a sarcoma than an epithelial tumor. I believe, however, that there are definite foci in this case where it shows up to be epithelial and that this range of spindle-cell differentiation is within the realm of possibility for a thyroid epithelial neoplasm. With the degree of capsular invasion we must conclude furthermore that it is a malignant neoplasm, and, therefore, I should like to call this an atypical carcinoma, perhaps arising from an atypical adenoma.

The relationship between the previous hyperthyroidism and the thyroid tumor is interesting for conjecture, but I am afraid that the relationship is difficult to prove. We have no way of knowing, for example, whether this tumor existed prior to the hyperthyroidism, even though the history did imply this. It is, of course, possible that the tumor was stimulated to more active growth with the episode of hyperthyroidism. But even in papillary carcinoma of the thyroid, where there seems to be the greatest possibility for cancer arising from hyperplasia, it is difficult to prove such a relationship in individual cases.

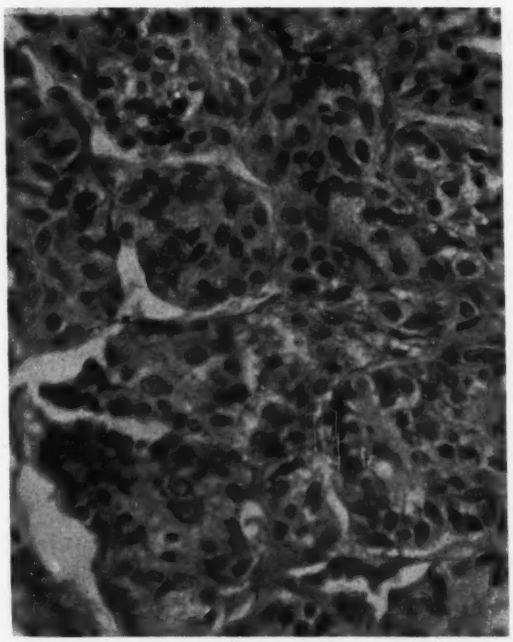

Fig. 13. Case 8. In a few areas the cells appear to be epithelial and tend to form follicles.

A tumor such as this is certainly undifferentiated as far as forming specific thyroid structures. However, the fact that it is undifferentiated does not necessarily imply that it is highly malignant. Tumors of this nature often seem clinically to be relatively low grade, and it is a mistake, therefore, to confuse undifferentiation with a high malignant potential.

Submitted Diagnoses.—spindle cell carcinoma, 19; carcinoma, 7; giant cell carcinoma, 3; giant and spindle cell carcinoma, 1; fibrosarcoma, 4; leiomyosarcoma, 1; spindle cell sarcoma, 1; neurogenic sarcoma, 1; neuro-fibroma, 2; cellular leiomyoma, 1.

DR. MEISSNER: Spindle cell carcinoma is a reasonable diagnosis. This is not the giant cell carcinoma as we usually see it. Although giant cell carcinomas of the thyroid also may have some cells of spindle shape, giant cell carcinoma of the thyroid is a highly malignant tumor and is composed of numerous giant epithelial cells of an obviously high order of malignancy. It is the giant cell carcinoma of the thyroid that is sometimes called carcinosarcoma.

True sarcomas of the thyroid do occur occasionally, but are extremely rare. Fibrosarcoma is the most common in this country. In Europe tumors called "hemangioendotheliomas" sometimes are seen in the thyroid. Apparently, we do not see them in this country or else we have a different terminology for them.

The problem of the classification of thyroid carcinoma sometimes is complicated by a variable structure of the tumor. Fortunately, this is not a common occurrence, but it is important to emphasize, as Smith[17] did many years ago, that before making the diagnosis of a sarcoma of the thyroid one should examine multiple sections to look for epithelial components. The thyroid cell has varying potentials in tumors; some tumors are highly vascular, some are papillary, some are follicular, some are epidermoid, and some appear lymphomatous. In a single case or even on a single slide of these tumors, depending on the area which one examines, it might be possible to make a diagnosis of vascular tumor, fibrosarcoma, undifferentiated carcinoma, follicular carcinoma, or papillary carcinoma.

We often are asked as pathologists why we have so few autopsies on patients with cancer of the thyroid, and whether this may not mean that few people die of their thyroid cancer. We recently reviewed the papillary carcinomas of the thyroid we had seen over a 20 year period. We found 278 surgical specimens over the 20 years, but in our autopsy files we had only three instances where patients died from their papillary carcinoma—not very impressive. We thought that maybe this wasn't too significant, however, unless it was compared with another type tumor, so we picked out cases of carcinoma of the larynx.

While we had 321 surgical specimens of carcinoma of the larynx over 20 years, we were amazed to find that there were four instances in our autopsy files where patients had died of their carcinoma of the larynx in the same 20 year period. I'm not sure what this proves, except that statistics can prove anything.

DR. WALTER KROHN, Galveston: Unfortunately, there is a misunderstanding in the terminology. You hesitate, Dr. Meissner, to call this one an undifferentiated carcinoma. Undifferentiated, in my understanding, means it is not differentiated into a pattern; whereas anaplastic refers to the individual cells. Unfortunately, in all textbooks anaplastic and undifferentiated are put in the same pot. A tumor can be highly undifferentiated, being not anaplastic; and a tumor can be well differentiated into a pattern, being terrifically anaplastic.

DR. MEISSNER: I agree, from the pathologic point of view I should like to call this an undifferentiated carcinoma; and yet, if we send that diagnosis back to our surgical colleagues, they will immediately think this is a highly malignant, wild cancer, which I don't think it is. I thought about this point last week and even noted that in Dorland's medical dictionary "anaplasia" is a synonym for "undifferentiated." It doesn't necessarily mean highly malignant, according to the dictionary, although I am sure most of us use the term anaplasia to signify a highly malignant tumor.

COL. LUKEMAN: After our surgical report went out the patient was transferred to Brooke Army Hospital where she was reexamined with the object of doing a radical neck resection. At the time she was examined no nodes were found, and I believe it was felt that further surgery was not indicated at that time.

COL. J. L. HANSEN, Brooke Army Hospital: No further surgery was done. To date she has no further evidence of disease.

Case 9 (Leiomyolipoma)

Diagnosis.—Leiomyolipoma.

Contributor.—Dr. J. H. Childers, the University of Texas Medical Branch, Galveston.

History.—This 55 year old colored female noted intermittent pain in the left lower quadrant of the abdomen for 1 year. A local physician told her she had a tumor of the uterus. During the last 3 months she had noted irregular vaginal bleeding. Grossly, the uterus, including the vaginal portion of the cervix, was 17 by 12.5 by 13 cm. An oval, soft, 12 by 14 cm. mass was present in the fundus and was surrounded by a 1 cm. rim of myometrium. The cut surface was yellow, greasy, and glistening. A second oval, 1.7 cm. mass was present in the myometrium that had a firm, gray-white cut surface.

DR. MEISSNER: Presumably the sections for the seminar were taken from the larger yellow myometrial mass. Microscopically it seems well demarcated from the adjacent normal-appearing uterine muscle. The mass is composed mostly of adult fat cells which focally show only slight degeneration.

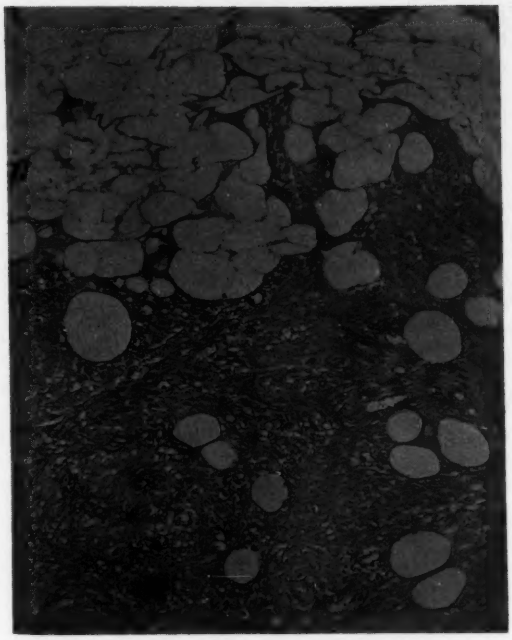

Fig. 14. Case 9. Leiomyolipoma of uterus. The tumor is vascular and composed of adult fat and smooth muscle.

Intermingled with the fat cells are varying-sized, irregular masses of cells of a different type. These cells are spindle shaped with spindle shaped nuclei and tend to be whorled and clustered about blood vessels, which are numerous. The spindle shaped cells, except for their whorled arrangement, appear quite similar to the cells of the adjacent normal myometrium. There is one mitosis, and cells and nuclei show little pleomorphism. The endothelial cells lining the numerous vascular spaces in these foci often are swollen and even occasionally suggest that they are epithelial rather than endothelial. Many of the vascular spaces contain blood.

I believe that this is a benign tumor composed of fat and smooth muscle and that, therefore, the best diagnosis is a leiomyolipoma.

Submitted diagnoses.—lipoma (fibrolipoma, 6), 18; leiomyolipoma, 7; angioleiomyolipoma, 2; hamartoma (fibroangiolipoma, 1), 3; benign mesenchymoma, 6; mixed mesodermal tumor, 1; liposarcoma, 1; leiomyosarcoma, 1; carcinoma of endometrium, 1.

The rather considerable number of small vessels intermingled with the smooth muscle in this lesion suggest the possibility of an angiomatous element. In fact, there are foci that somewhat resemble the

so-called genital mesothelioma or lymphangioma. The vascular components, however, do not seem to me to be well enough developed to consider this diagnosis, and the inclusion of so much fat within the tumor also would be unusual. However, it is quite possible to think of this tumor as arising from embryonic multipotential components and in this sense comparable to the genital mesothelioma. There are some who feel that most smooth muscle tumors of the uterus arise from primitive cells, and it, therefore, is not surprising that one occasionally sees a mixed tumor such as this. Cases of pure lipoma have been reported as occurring within the uterine musculature.

The proliferation of smooth muscle around vascular spaces is also of interest since another theory concerning the development of leiomyomas is that some of them begin not from the uterine musculature itself but from smooth muscle of vascular walls.

DR. LEO LOWBEER, Tulsa: I think this tumor is identical with those found in the kidneys. Three years ago Dr. Dockerty presented one like it before this group. In our laboratory, we recently saw a huge tumor of the kidney in which both the myomatous and the lipomatous elements had become sarcomatous.

DR. F. P. BORNSTEIN, El Paso: We just had a large yellow tumor, and I couldn't decide whether it was a myosarcoma or a liposarcoma; there seems to be some basic relationship between those tissues as they appear in the uterus.

DR. MEISSNER: The tumors that occur in the kidney in tuberous sclerosis are similar to this tumor —a mixture of fat, smooth muscle, and blood vessels.

Case 10 (Mycosis Fungoides)

Diagnosis.—Mycosis fungoides with reticulum cell sarcoma.

Contributor.—Dr. Meissner.

History.—A salesman, aged 53, with a history of repeated attacks of eczema as a child, developed an erythematous, scaly rash at age 48. Over the next several years he received several courses of spray x-ray and finally cathode ray therapy, but the rash persisted over the entire body. A few months before death cutaneous and subcutaneous nodules of varying size appeared and his condition steadily deteriorated. Blood counts were normal until 1 month before death when the findings were: hemoglobin 6.7 Gm., red blood count 3,370,000, and white blood count 2,450 with differential of neutrophils 52 per cent, bands 29 per cent, lymphocytes 4 per cent, monocytes 6 per cent, and immature (including 1 per cent blasts), 9 per cent.

DR. MEISSNER: This 53 year old man had a 5 year history of skin lesions and numerous complications—too numerous to give in detail in the history. Five years before death a biopsy of the skin was diagnosed as mycosis fungoides. He developed progressive skin lesions which were sometimes temporarily held in check by radiation, and ultimately was seen at our hospital for cathode ray therapy. This is a superficial type of radiation which can be given as total body radiation. It has a penetrating distance of millimeters rather than centimeters and is, therefore, adaptable for the treatment of superficial skin lesions. This man responded well for a while, as do many patients with mycosis fungoides or skin lymphoma, but he later had a rapid downhill course with numerous tumor nodules in the skin.

Fig. 15. Case 10. Mycosis fungoides with reticulum cell sarcoma. Skin showing ulceration and a cellular infiltrate.

Microscopically, section of the skin removed at autopsy showed an infiltrate of pleomorphic cells with many atypical forms and numerous mitoses. I should add that silver stains showed a fine reticulum about most of the cells. The epidermis is ulcerated and necrotic. Perhaps before giving my diagnosis and follow-up information from the autopsy, I should ask for the tabulated diagnoses.

Submitted diagnoses.—mycosis fungoides, 16; malignant melanoma, 6; Hodgkin's disease, 5; reticulum cell sarcoma, 5; myeloid leukemia, 5; monocytic leukemia, 1; plasma cell myeloma, 1; Weber-Christian disease, 1; Kaposi's disease, 1.

DR. MEISSNER: At autopsy, in addition to multiple skin lesions, such as we have already seen, there were similar tumor nodules involving the heart, lungs, tongue, spleen (340 Gm.), pancreas, liver (2,300 Gm.), adrenals, kidneys, testes, thyroid, parathyroids, bone marrow, and lymph nodes of the neck, axilla,

mediastinum, and abdomen. He died with terminal bronchopneumonia and uremia. We interpreted this as being a widespread reticulum cell sarcoma of a malignant sort, which some might call Hodgkin's sarcoma.

This case represents what was clinically mycosis fungoides and what has ended up pathologically as a highly malignant disseminated lymphoma, involving essentially all parts of the body.

During the past few years, because of patients being referred for cathode ray therapy for the treatment of clinical mycosis fungoides, we have seen a considerable number of biopsies of the skin of such individuals. The experience from reading these biopsies has led us to feel that while mycosis fungoides is a reasonable clinical entity, it may be extremely difficult to diagnose as a pathologic entity. The reason for this is that the disease known clinically as mycosis fungoides characteristically goes through several stages. In the first stage, which is known to the clinicians as erythematous or premycotic state, a biopsy of the skin usually shows minimal changes of chronic, nonspecific dermatitis. The round cell infiltrate is a little deeper than one usually sees in ordinary dermatitis, but the picture is not diagnostic of a lymphoma and certainly not of a specific lymphoma. As the disease progresses clinically it enters what is called the plaque stage, at which time a biopsy may be diagnostic. In this stage there is frequently a pleomorphic cell infiltrate at the corium which may resemble somewhat a Hodgkin's disease infiltrate, and there are often small microabscesses in the overlying epidermis which are called Pautrier abscesses. This picture may be specific enough to suggest strongly the diagnosis of cutaneous lymphoma and, more particularly, mycosis fungoides. The third clinical stage is known as the nodular or tumor stage, and here the biopsy shows the full-blown changes of lymphoma cutis, at times resembling Hodgkin's disease, reticulum cell sarcoma, or other lymphoma. During all of these stages the clinical diagnosis may be mycosis fungoides, but obviously the pathologic diagnosis is possible in only a limited number of cases.

Many pathologists have felt that mycosis fungoides is only a manifestation of cutaneous lymphoma and, therefore, should not be considered as a separate pathologic entity. This may, in part, be true from the pathologic point of view, but it is not true from the clinical point of view. To the clinician mycosis fungoides is an entity usually distinct from other types of lymphoma. It is true that many patients with mycosis fungoides ultimately show disseminated lymphoma such as in the seminar case here; and, further, this disseminated lymphoma may be similar to a lymphoma unassociated with mycosis fungoides.

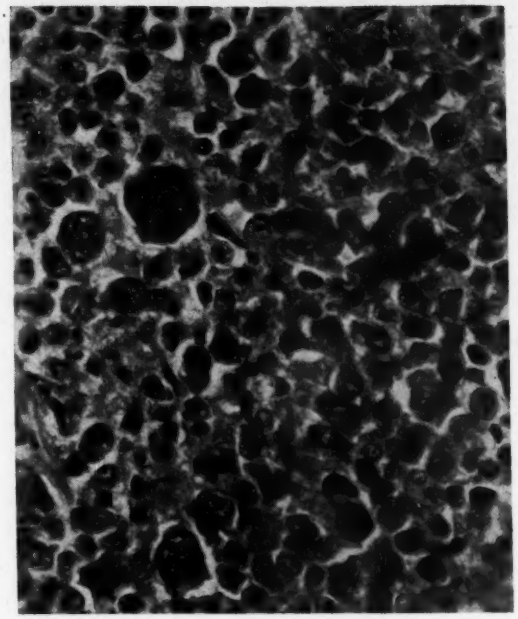

Fig. 16. Case 10. The infiltrate is pleomorphic.

Gall[4] in a review of the autopsies of 129 cases of mycosis fungoides reports that the ultimate lesion, whether neoplastic or not, is confined to the skin in 39 cases, and further, that at the termination of the disease there was recognizable malignant lymphoma of one form or another in two-thirds of the cases. Our current feeling about the term mycosis fungoides is that while it is a useful one to the clinicians, it is a difficult one in most instances for the pathologist to use as a specific diagnosis.

DR. GEORGE MANI, San Antonio: This is mostly a question in terminology. In view of the hematological findings terminally, why couldn't you call this some form of a reticuloendotheliosis—in other words, a leukemia, a terminal leukemia, rather than a lymphosarcoma?

DR. MEISSNER: I would have no objection to that diagnosis. This man at least had a leukemoid picture. I'm not sure that he had a leukemia although the bone marrow was massively involved by infiltration with the tumor. I can go along with Custer's[1] views about the intermixture of all of these lymphomas, and how they change from one type to another. So to have leukemic manifestations in a patient with reticulum cell sarcoma seems perfectly reasonable to me, and I wouldn't object if one wanted to call this leukemia in addition to the other findings.

DR. SEVERANCE: Were these immature cells possibly just tumor cells circulating in the blood?

DR. MEISSNER: Yes, I think they may well have been just tumor cells, but does that necessarily make it a leukemia? I'm not sure whether it does or not.

Case 11 ("Desmoid" Tumor)

Diagnosis.—"Desmoid" tumor.

Contributor.—Dr. A. O. Severance, Baptist Memorial Hospital, San Antonio.

History.—A 33 year old female noticed a slightly painful enlargement just medial and next to the spine of the left iliac crest. This was first noted while taking exercises after the birth of a child 2 years before. She had become conscious of it, but did not pay any attention to it until recently. X-ray showed it to be a soft-tissue mass, not connected with bone. Examination of the patient's back showed a small mass medial and next to the spine of the left iliac crest, appearing to be connected with the fascia over the crest of the ilium. It was slightly tender and freely movable. The specimen consisted of a rounded mass measuring 4 by 5 by 3.5 cm. It was firm, pinkish-gray, and well circumscribed. The cut surface was pink and glistening with grayish fibrous bands interdigitating in all directions.

DR. MEISSNER: This 4 cm. circumscribed mass which had been present for at least 2 years was removed from the subcutaneous tissue of the lower back. Microscopically it is composed primarily of spindle shaped cells which contain spindle shaped nuclei without mitoses. In some foci there is little variation either in the cells or their nuclei, and between the cells are strands of collagenous tissue which are intimately associated with the outlines of the cells. In other foci the cells show more variation in size and shape with some stellate forms; here the nuclei are also more variable with some of large size but still without mitoses. In these foci the cells are in a looser arrangement, and are less intimately associated with collagen fibers. Both the connective tissue fibers and the cells tend to grow in an interlacing, often whorled pattern but without definite structural formation. At the periphery of the mass some of the cells with their collagenous fibers extend as an infiltration into the adjacent striated muscle. The stroma shows moderate vascularity.

The problem in this case is largely a matter of nomenclature. The spindle shaped cells are forming collagen and are therefore fibroblasts. This, then, is a lesion composed of proliferating fibroblasts or fibrocytes, which is infiltrating or invading adjacent striated muscle.

I know of no certain way to determine whether or not this lesion is even a true tumor. Dr. Stout[19] makes a particular point of the difficulties of determining if there is such a thing as a benign tumor composed of fibroblasts.

About the only thing of which we can be certain here is that this is a mass of proliferating fibroblasts. The name for such a lesion is always a difficult problem for the pathologist. It is my feeling that a lesion such as this should best be given a descriptive diagnosis, such as low-grade proliferation of fibrous tissue. We know, however, that in the practice of pathology such a diagnosis is not readily acceptable to our clinical colleagues. They want us to put a name to the process and to tell them whether it is a benign or malignant tumor.

Fig. 17. Case 11. "Desmoid tumor." Proliferating spindle-shaped fibroblasts growing in an interlacing, often whorled pattern.

To call this an unqualified fibrosarcoma is somewhat of an overdiagnosis in spite of the fact that the tumor is extending into adjacent muscle. A few years ago, Musgrove and McDonald wrote an interesting paper[13] on extra-abdominal desmoid tumors, and if one must apply a specific name to this lesion I think desmoid tumor is as reasonable as any, for several reasons. First, the tumor has occurred in a female following pregnancy; this is a common history in desmoid tumors. Second, the tumor has striated muscle fibers included within it, again a common finding in desmoid tumors. The low-grade nature of the fibrous proliferation is also consistent with this diagnosis.

My diagnosis on the lesion, then, is "desmoid" tumor.

Submitted diagnoses.—desmoid tumor, 13; fibrosarcoma, 7; leiomyosarcoma, 4; rhabdomyosarcoma, 1; fibroma (neuro- 1; fibromyxoma, 1), 7; neurilemmoma, 2; fibromatosis, storiform, 1; fibromatosis, nodular, 1; "milk-line" fibroadenoma, 1; hemangioendothelioma, 1; malignant giant cell tumor, 1.

DR. MEISSNER: There is no more difficult field for pathologic nomenclature than that of fibrous tissue proliferations of this general type. We, as pathologists, all know how difficult it may be to distinguish, for example, between scar tissue and a

keloid, or even between scar tissue and low-grade fibrosarcoma. Unfortunately, in many instances, the only way the diagnosis can truly be determined is by following the clinical course and noting whether or not recurrences or metastases occur.

It is quite possible that this lesion represents a very low-grade fibrosarcoma. I do not think so, but on the other hand, I have seen metastasizing fibrosarcomas which were at least focally as well differentiated as this lesion.

Musgrove and McDonald in their paper made a particular point of attempting to distinguish between extra-abdominal desmoid tumors and true fibrosarcomas. They found, as might be expected, that many fibrosarcomas could be easily distinguished from desmoid tumors by the presence of numerous mitoses, tumor giant cells, considerable pleomorphism of cells, and a high degree of cellularity. They were careful to point out, however, that the differential diagnosis between a fairly cellular, benign, fibrogenic lesion and a low-grade fibrosarcoma is one of the most difficult decisions a pathologist must make. The only way the pathologist can be sure, and not 100 per cent at that, is to determine the final outcome in each individual case.

It always has seemed unfortunate to me that our nomenclature of tumors must emphasize the distinction between benign and malignant. It is true, of course, that such a distinction often can be made quite readily; but in tissues which are as uncomplicated as connective tissue, criteria for distinguishing not only between benign and malignant tumors, but also between simple reactive proliferation and tumor are often lacking.

DR. SEVERANCE: This patient was last seen by the family doctor about the middle of August of this year (1958). In the interval since 1952, when this lesion was removed, the patient has had one more successful pregnancy. There have been no additional lumps or bumps of this type, and the area from which this was removed is still free of any recurrence of anything that can be felt clinically.

DR. SMITH, Lakeland: Did you think that histologically they got around the tumor?

DR. SEVERANCE: That's always a good question. I think they had a small amount of fat around the tumor.

DR. A. M. RICHMOND, San Antonio: Dr. Meissner, has it been your observation that this type of tumor in the neck region is apt to look much more malignant than it will eventually act?

DR. MEISSNER: I don't know about the tumor in the neck region specifically as looking more malignant; I think all of these lesions tend to look more malignant than they act. They rarely metastasize although it is true that they may recur locally, and they may recur to such a degree that one might think of them as being locally malignant.

DR. J. J. ANDUJAR, Fort Worth: This is, as Dr. Meissner says, an exercise in semantics rather than pathology, so I would like to ask Drs. Severance and Meissner why they use the word extra-abdominal desmoid tumor. There is ample precedent for stating that desmoid can occur in numerous other areas, including the neck, so why tack on extra-abdominal?

DR. MEISSNER: I don't think one needs it. Actually, the definition of desmoid tumor is a fibrous tumor, or fibrous proliferation, not necessarily a tumor; and the dictionary definition of a desmoid tumor does not specify that it must be in the abdominal wall.

DR. SEVERANCE: Don't you imagine that the first attention called to these tumors was to those happening to be in the abdominal region following pregnancy, and that is the reason everybody talks about extra-abdominal when they occur elsewhere?

DR. C. P. SCHWINN, Houston: We had a fair number of these that fit the classification that McDonald set out, and we have seen them at the M. D. Anderson Hospital and Tumor Institute sometimes in their second, third, or fourth recurrence. We have found that none of them have ever metastasized, but that they frequently will grow along fascial sheaths, nerves, and so on, so that in a few cases amputation has been necessary.

Case 12 (Malignant Giant Cell Tumor)

Diagnosis.—Malignant giant cell tumor of soft tissue.

Contributors.—Drs. D. A. Todd, A. M. Richmond, and Sylvia Johns, Nix Hospital Clinical Laboratory, San Antonio.

History.—This 58 year old colored female first noticed a "swelling" in the left antecubital fossa early in 1956, which was said to have grown rapidly. The mass was removed but apparently no tissue studies were done. The growth rapidly recurred, and in May, 1956, several circumscribed, lobular, solid, yellowish brown tissue masses were removed, the largest 13 by 25 mm. in size. In July, 1956, the patient was referred to a hospital because of pain in the right elbow; at this time the antecubital fossa was explored, and a nodule was "removed from the ulnar nerve." The tissue for examination now is that which was removed in May, 1956. Past history indicated a "stroke" in 1953, positive serology, and moderate hypertension.

DR. MEISSNER: From the history on this case we find that the tissue represents a rapidly recurring growth removed from the region of the elbow. We are not told whether the original growth involved bone or not, but we are sure that the recurrence was removed from soft tissue.

Microscopically the tissue is composed primarily of various sized masses of uniform-type structure embedded in fibrous tissue and muscle. These masses are composed of spindle, ovoid, and round cells which

separate and surround giant cells. The giant cells often contain many nuclei and tend to be more numerous near blood vessels. Some, in fact, seem to be lying within vascular spaces. While the cytoplasm of the giant cells shows an occasional vacuole, these are not numerous and no foreign material is found. The smaller cells with single nuclei nevertheless have nuclei similar to that seen in the giant cells. An occasional one of the smaller cells shows a mitosis. About the periphery of the masses of cells, there is some fibrous tissue and muscle which is intimately intermingled with the cells just described. In the adjacent fibrous tissue there are small nests of identical cells present. The stroma shows focal deposition of hemosiderin pigment; there is no new bone or cartilage formation.

This is a lesion composed predominantly of giant cells, and my first decision is to call this a giant cell tumor. To me it appears quite comparable to the giant cell tumor of bone. It has less resemblance, because of its lack of xanthomatous stromal change, to giant cell tumors arising in tendon sheath. I think, however, that is also a possibility.

The next decision pertains to whether or not the tumor is malignant or benign. Certainly from the morphology alone one does not get the impression of a malignant tumor. The lesion is actively proliferating, however, and there are numerous foci of tumor cells growing in connective tissue, which is something that a benign giant cell tumor is not supposed to do. Also, from the history we know that this lesion has recurred promptly and is involving such important structures as the ulnar nerve.

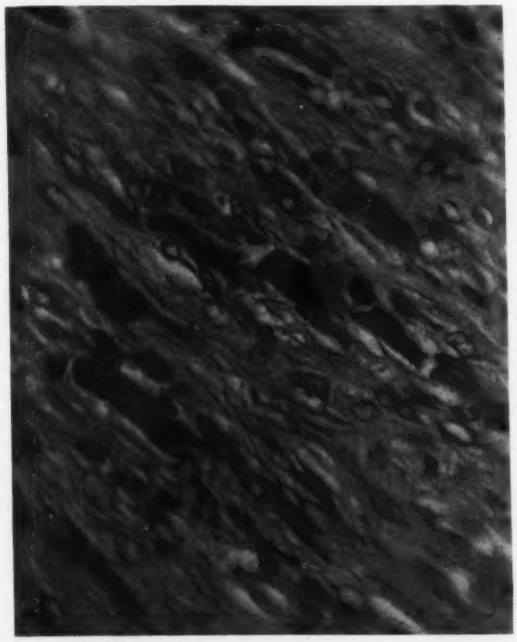

Fig. 18. Case 12. Malignant giant cell tumor. Sheets of round, ovoid and spindle cells surround and separate numerous multinucleated giant cells.

I believe, therefore, that we must consider this at least a locally malignant tumor of giant cells which may even have the capability of metastasis. My diagnosis, therefore, is malignant giant cell tumor of soft tissue.

Submitted Diagnoses.—giant cell tumor, recurrent, 15; angiosarcoma, 3; synovial sarcoma, 3; liposarcoma, 1; rhabdomyosarcoma, 1; hemangioendothelioma, 6; hemangioma (with giant cell reaction, 2), 3; neurofibroma, 2; myxofibroma, 1; pigmented villonodular synovitis, 2; malignant ganglioneuroma, 1; malignant tumor, 2.

DR. MEISSNER: Giant cell tumor, recurrent, does not indicate whether it was thought to be benign or malignant, but that is a good point we can discuss in a moment.

There are actually three decisions to be made in discussing this case. First, is this lesion a tumor, or is it merely reaction? We know of no history of trauma, but it is true that the previous surgery may have stimulated or disseminated the process so that it now seems more like tumor. This decision is further complicated by the fact that there is no universal opinion as to whether giant cell lesions are pure neoplasms anyway. There are those who feel that some of them at least represent merely reactions to injury, or that the giant cells are accompaniments of other basic nontumorous or tumorous processes.

The second decision deals with site of origin of a lesion such as this. I believe this tumor is quite comparable to many of the giant cell tumors arising in tendon sheath. These latter tumors, however, almost always occur in the hands or feet; this would be an unusual site. Furthermore, there are not the extensive xanthomatous changes that one frequently finds in such lesions. Recurrences of giant cell tumors of tendon sheath are uncommon but do happen. I believe that a lesion such as this might arise from other soft tissue, perhaps synovia, muscle, and blood vessels. I have seen several cases where there have been such accumulations of giant cells associated with thyroid carcinoma, but not apparently an inherent part of the carcinoma. Rather[15] believes that giant cells of this type, which are variously called osteoclasts, foreign body giant cells, and so forth, may arise from the endothelial lining of vessels.

The third consideration regards the question of benign versus malignant tumor. Here again the decision is a difficult one—recurrence of benign giant cell tumor is uncommon if it has been completely excised. In favor of a malignant process in this case is the history of rapid growth, rapid recurrence, and involvement of such important structures as the ulnar nerve. The dissemination of the lesion through-

out the fibrous stroma is suggestive to me of malignant nature. Mitoses are fairly numerous in this case, as they are in malignant variants of giant cell tumor; but, again, they may be seen in benign tumors as well, and this certainly is not a definitive criterion. The decision as to whether a giant cell tumor is benign or malignant is extremely difficult, and only the clinical course can give the true answer.

This particular tumor, I believe, has the potentials of a malignant one. I have seen cases of giant cell tumor which have metastasized which have looked no worse than this.

DR. LEO LOWBEER, Tulsa: Couldn't we seriously consider that this is a tumor or tumorlike hyperplasia of synovial cells, and not from a joint, and not from tendon sheath, but from the antecubital fossa? Synovial cells are notorious for acting as macrophages, storing lipid or hemosiderin, or fusing to form giant cells. Some of the lesions in the joints, like villonodular synovitis, behave and look like tumors and are not, so perhaps this is a tumor originating from the antecubital fossa.

DR. MEISSNER: I think it is possible that this may have arisen from synovia. I would place that among the first of the possibilities as the site of origin.

DR. RICHMOND: The surgeon in this case said that the bone was not involved at any time so far as the x-rays were concerned. She was operated upon the last time in July of 1956, and in December of 1957, she was apparently free of disease.

Case 13 (Undifferentiated Sarcoma)

Diagnosis.—Undifferentiated sarcoma, probably rhabdomyosarcoma.

Contributor.—Dr. J. M. Moore, Santa Rosa Hospital, San Antonio.

History.—A small, painless mass was noted in the calf of the leg of a 1½ year old male. At this time it was the opinion of the family physician that it was a cyst, and he advised awaiting further developments. The mass gradually grew for 1½ years and was then excised locally. The tumor measured 8 by 5 cm. and was somewhat irregular, lobulated, and fairly well circumscribed. Upon sectioning it was found to be practically solid in consistency, with a homogeneous appearance. There were also inguinal nodes revealing tumor metastasis. The tumor was thought to be embedded in muscle and not attached to bone. Cobalt 60 radiation is being given at this time.

DR. MEISSNER: This tumor was removed from a young child, and at the time of removal there already were metastases. It apparently arose in muscle.

I think we can all agree that this is a malignant tumor, both from the proof provided from lymph node metastasis and by the extensive invasion of adjacent structures and blood vessels. Furthermore,

Fig. 19. Case 13. Undifferentiated sarcoma, probably rhabdomyosarcoma. In many foci the tumor cells show a tendency to grow in clusters.

the tumor does not structurally suggest the possibility of either a lymphoma or a carcinoma, so the problem, then, is to give this sarcoma a name.

To classify sarcoma one can utilize characteristic structural arrangements, the formation of recognizable material by the tumor cells, and the appearance of the tumor cells.

One of the striking features of this case is the tendency of the tumor cells, in many foci, to grow in clusters. This alveolar arrangement suggests the diagnosis of alveolar soft part sarcoma, which is also sometimes called malignant granular cell myoblastoma and nonchromaffin paraganglioma. This diagnosis is perhaps an acceptable one since this is a sarcoma of soft parts and it is partly growing in alveolar arrangement. These tumors characteristically have larger cells, however, often with granular cytoplasm, and they contain few or no mitoses. These characteristics are not present in our tumor.

In other foci of the tumor the cells are intimately associated with collagen, strongly suggesting that the collagen is being formed by tumor cells. This tumor, therefore, might be considered a variant of fibrosarcoma.

In still other foci, some of the tumor cells are vacuolated, and although no material is available for a fat stain, the vacuoles could be fat and the diagnosis of liposarcoma is, therefore, a possible one.

In still other foci, there are tumor cells which have

a deeply acidophilic cytoplasm and in these foci there are, at times, elongated cells strongly suggesting striated muscle cells. I looked for a long time for cross striations but was unable to find any.

This, then, is a tumor with a variable pattern and perhaps a variable cell type, and I suppose that the best term is a noncommittal one such as undifferentiated sarcoma. I should like very much to call this a rhabdomyosarcoma, but in the absence of cross striations, I do not feel quite justified in doing so.

Submitted Diagnoses.—rhabdomyosarcoma, 7; rhabdomyosarcoma, embryonal type, 10; soft part alveolar sarcoma, 3; synovial sarcoma, 4; myxosarcoma, 1; reticulum cell sarcoma, 1; angiosarcoma, 1; liposarcoma, 1; sarcoma (type?), 2; hemangioendothelioma, 3; leiomyoma, 1; fibroma, 1; neurilemoma, 1; hemangio-pericytoma, 1; carcinoma of sebaceous glands, 1.

DR. MEISSNER: Obviously there is a wide variance of opinion as to the proper name for a sarcoma such as this.

The possibility of rhabdomyosarcoma is still intriguing. Dr. Horne[7] has described an alveolar arrangement in some rhabdomyosarcomas and perhaps the alveolar arrangement in this case can be explained by this diagnosis. Primitive striated muscle tumors may have a varied appearance and at times may even resemble lymphomas. The necessity for careful search for typical striations must be emphasized.

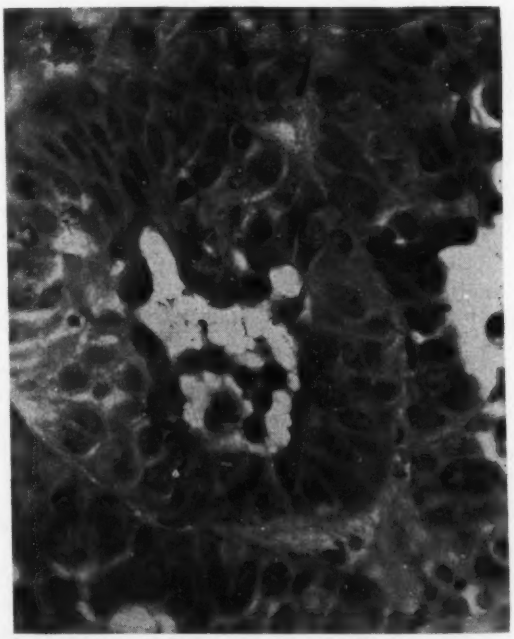

Fig. 20. Case 14. Low-grade carcinoma of laryngeal glands. Large epithelial cells form alveolar structures.

The metastasis to lymph nodes, by a sarcoma, is unusual in adult sarcomas, but is more common in sarcomas of children. In 23 cases of rhabdomyosarcoma which Horne was able to follow adequately, 15 showed metastases to lungs, but 13 showed metastases to lymph nodes.

DR. GEORGE MANI, San Antonio: Did you at any time consider calling a tumor a mesenchymoma?

DR. MEISSNER: Yes, I think that could be my best diagnosis, a malignant mesenchymoma, if I didn't find any cross striations. If I had to call it a specific type, I would call it a rhabdomyosarcoma.

DR. MANI: How many components are needed before calling a tumor a mesenchymoma?

DR. MEISSNER: Dr. Stout is the one who invented this word, and I don't know—maybe someone else here does—exactly what his necessary number is; I suppose that three or more components should be present.

DR. SEVERANCE: I would have said he considered two or more, but he doesn't include the fibrous tissue element.

Case 14 (Low-Grade Carcinoma of Laryngeal Glands)

Diagnosis.—Low-grade carcinoma of laryngeal glands.

Contributor.—Capt. F. W. Kiel, M.C., Brooke Army Hospital, Fort Sam Houston.

History.—A 68 year old white male with a history of a squamous cell carcinoma of the floor of the mouth treated surgically 4½ years previously complained of dysphagia of 2 months' duration. There was no voice change nor difficulty with respiration. A pedunculated lesion with a very thin one-half inch stalk, attached in the region beneath the outer arytenoid, was removed. The tumor was bluish, firm, and somewhat nodular, measuring generally 1.6 cm. The cut surface was dark red.

DR. MEISSNER: This patient had a history of previous removal of a squamous cell carcinoma, grade one, from the floor of the mouth. The lesion presented for the seminar was pedunculated and attached to the left false vocal cord and arytenoid. I do not believe there is a relationship between the squamous cell carcinoma of the floor of the mouth and the lesion we have here.

The lesion is composed of more or less circumscribed clusters of cells which underlie stratified squamous epithelium. The typical cell type is tall, cuboidal, contains a large nucleus, and appears seated on a basement membrane. The opposite side of the cell is directed toward a lumen space. The cell nucleus is large. Mitoses occasionally are found. The cytoplasm of the cell is pale or slightly acidophilic. There are not granules or droplets.

There are numerous variations from the typical cell apparently due to cell distortion or to cell degeneration. At times the cells are rounded and at

other times greatly elongated, but all of these seem to be merely variants of the basic cell component. For the most part the cells tend to form an alveolar structure, but at times they are in more solid masses. There is no definite keratin formation, no intercellular bridges that I could find, and no formation of mucus.

The cells, where they are adjacent to the stratified squamous epithelium, show a minimal invasion. The cells also are invading stroma, and there are numerous clusters in and lining vascular spaces.

The stroma for the most part is scanty but highly vascular. In a few areas it has a hyaline appearance, but there is no formation of cartilage. At one point near the periphery of the tumor are several normal-appearing ducts, and the appearance suggests that these are leading from the epithelial surface into the tumor.

The differential diagnosis in this case includes many possibilities, such as metastatic carcinoma, malignant melanoma, hemangioendothelioma, and so forth. Rather than discuss all of these, I should like to point out the features in this lesion which have led me to my diagnosis. In the first place, these cells look to me as though they are epithelial. The tall, cuboidal nature and the tendency to grow in clusters with some alveolar arrangement are characteristics of an epithelial neoplasm. Secondly, the tumor cells are invading stroma, blood vessels, and overlying squamous epithelium; therefore, this is a malignant epithelial neoplasm.

The site or origin of these cells is an interesting subject for conjecture. The stroma is not very typical of mixed tumor of salivary gland, and yet there is a suggestion here and there of the dense hyaline material that often is seen. The presence of several duct structures which seem to lead into the tumor suggests further that this is a tumor arising from accessory mucous or serous glands of the larynx. The acidophilia of some of the tumor cells resembles somewhat that of the so-called oncocytes seen in some salivary gland lesions. The absence of convincing keratinization and of mucus is disappointing, but nevertheless, both of these may be absent in salivary gland tumors. From several points of evidence, then, none of which is in itself conclusive, I believe this tumor is a low grade carcinoma arising from laryngeal glands. I think it also may be considered a carcinoma of accessory salivary gland tissue.

Submitted Diagnoses.—squamous cell carcinoma, 13; metastatic squamous cell carcinoma, 8; adenocarcinoma, 2; malignant melanoma, 4; hemangioendothelioma, 4; adenoma, mixed tumor type, 3; salivary gland adenoma, 2; adenoma, 1; glomangioma, 1; hemangiopericytoma, 1; paraganglioma, 1.

DR. MEISSNER: The people who called this a squamous cell carcinoma apparently are in the majority. I just could not convince myself that any of the pink material I saw was keratin. If I had found keratin, however, I would just include the tumor in the group of salivary gland neoplasms where there is an epidermoid component as well as the glandular component.

Fig. 21. Case 14. There is invasion of the overlying squamous epithelium.

Malignant melanoma is always a possibility in a tumor that is as difficult to classify as this, and the same is true of hemangioendothelioma. I must confess that I have never seen any tumor exactly like this one before. As one studies a series of salivary gland tumors, however, he is impressed by the great diversity in possible morphology. This is true not only in the mixed tumor group, but also in other forms of salivary gland tumors, both benign and malignant.

DR. R. P. BERNSTEIN, El Paso: There is one point I definitely noticed about this tumor, and that is that while the cells look very much like epithelial cells, they seem to be oriented in a most peculiar way toward blood vessels.

DR. MEISSNER: The relationship to the blood vessels bothered me quite a bit, but I could not think of any vascular cells that could look so epithelial.

DR. SEVERANCE: In the Atlas of Otolaryngic Pathology by Ash and Raum, mention is made of tumors which look like adenomas of the bronchus but are found in the larynx. Maybe one does not have to be too dogmatic and say whether it is benign or malig-

nant, because adenomas of the bronchus often behave as malignant tumors, even though they do not look as if they were.

DR. MEISSNER: I have never seen a bronchial adenoma that looked quite like this, but I would not be surprised if this is not a first cousin to bronchial adenoma.

DR. KIEL: After 1½ years this man has no symptoms, and examination shows a small .3 cm. papule in the operative site which was considered too small to biopsy. The clinical impression was possible minimal recurrence.

Case 15 (Mixed Carcinoma of Pancreas)

Diagnosis.—Mixed carcinoma of pancreas.

Contributor.—Dr. D. L. Galindo, Robert B. Green Memorial Hospital, San Antonio.

History.—This 22 year old Latin-American female with the chief complaint of epigastric discomfort, including some nausea, vomiting, and abdominal pain, had a cholecystectomy on August 29, 1957. At that time a cystic mass was demonstrated in the head of the pancreas. Roentgenograms revealed a partial distortion of the second part of the duodenum and the region between the duodenal bulb and the ampulla of Vater also was represented by a narrowed column of barium in which the mucosal pattern was edematous. On November 14, 1957, the tumor of the head of the pancreas was resected. At the time of operation no obstruction of the pancreatic or common bile duct system was demonstrated.

DR. MEISSNER: The tumor removed from the head of the pancreas is said to be cystic. This is not apparent from the microscopic section, but it is apparent that the growth is an epithelial one and that it is in part papillary. The papillary arrangement of the cells is not the same as the serpentine arrangement that one sees in many islet cell tumors. In some foci the cells are growing in rather solid masses. The individual cells are sometimes cuboidal, or even columnar, and other times rounded and with indistinct cytoplasmic outlines. Some of the cells suggest a fine vacuolization but mucous stains are negative. In one portion of the tumor there is a field showing a duct-like structure adjacent to cells which are highly suggestive of islet cells. The stroma in many parts of the tumor shows small foci of hyalinization which is something commonly seen in functioning islet cell tumors. There is also obvious stromal invasion, so the tumor is definitely malignant.

This is a malignant epithelial tumor with elements suggesting duct and islet cells and perhaps also some suggesting acinar cells, although there are no granules in the cytoplasm and there is not the usual necrosis that one finds in the typical acinar-cell carcinoma of the pancreas. I believe that this is a carcinoma of the pancreas which is low grade and papillary and which is composed of several elements of the pancreas, mostly primitive and poorly developed —a mixed carcinoma.

Submitted Diagnoses.—islet cell adenoma, 22; papillary cystadenoma, 8; papillary adenocarcinoma, 4; ductal cell carcinoma, 1; adenocarcinoma, 2; glomus tumor, 2; hemangiopericytoma, 1.

A few years ago, Sommers and I[18] reviewed 142 autopsy cases of pancreatic carcinoma. We were surprised not only by the diversity of types, but also by the range of unusual growth patterns. A mixture of islet cells with acinar or duct cells is much more common in pancreatic carcinoma than is usually appreciated. It also seems that we may overlook many nonfunctioning islet cell tumors because of their intermingling with other epithelial elements of the pancreas. Included among the unusual pancreatic tumors that we found were adenocanthoma, ciliated carcinoma, papillary carcinoma, and carcinoma resembling sarcoma.

The presence of hyalin in the stroma of an islet cell tumor is of considerable interest because this seems to be present only in the functioning islet cell tumors. This hyalin appears quite similar to the hyalin that is present in the islets of many diabetic patients. There is a small amount of hyalin in this tumor, and I think it is possible that the tumor in part represents an islet cell tumor which is functioning to a low degree.

DR. GALINDO: This patient has become pregnant

Fig. 22. Case 15. Mixed carcinoma of pancreas. Papillary area with a focus of stromal hyalinization.

since she left the hospital and to date, after 1 year, has not had any more symptoms.

DR. LEO LOWBEER, Tulsa: Have you ever seen carcinoids of the pancreas?

DR. MEISSNER: I have never seen one arising in the pancreas itself. I have, however, seen one in the ampulla of Vater.

DR. GEORGE MANI, San Antonio: Could you give us an idea about what percentage of pure islet cell adenomas or carcinomas are nonfunctioning?

DR. MEISSNER: I think the majority of islet cell tumors are nonfunctioning, but we don't recognize them, because we don't think about looking for islet cells unless the tumor is functioning. If a surgeon takes out a carcinoma of the pancreas, we don't usually think of the possibility of there being islet cells in that tumor unless it was a functioning tumor clinically.

DR. MANI: I believe there are some fairly specific stains for islet cells. Have you applied any histochemical tests to these tumors to see if those are actually islet cells?

DR. MEISSNER: Yes, but stains for islet cells in tumors are still not completely satisfactory. Unless the islet cell tumor is placed in fixative very soon after removal, it is difficult to do definitive islet cell stains.

DR. J. H. CHILDERS, Galveston: Do you feel that the differential staining of alpha cells offers any practical importance as far as correlative studies of hyperplasia in connection with ulceration of the gastrointestinal tract?

DR. MEISSNER: I haven't seen, in my own experience, a convincing case of ulceration caused by a proliferation of alpha cells.

DR. T. R. SUNBURY, Temple: In your review of pancreatic tumors, do you find that they carry the ductal and islet components as metastases?

DR. MEISSNER: Yes, these tumors do. They seem to grow with the same potentials in the metastases as in the primary, and I think once in a while that may give one a suggestion, at least, as to a primary site in the pancreas.

DR. J. J. ANDUJAR, Fort Worth: Do you recognize the term "nesidioblastoma," and if so, do you use it to indicate the nonfunctioning, highly anaplastic islet cell tumor?

DR. MEISSNER: We haven't used that term for islet cell tumors. We just call them islet cell tumors, benign, malignant, or questionable.

DR. J. E. WILLIAMS, Abilene: Isn't it true that often these adenomas are multiple and that sometimes they may occur outside of the pancreas?

DR. MEISSNER: Islet cell tumors—both functioning and nonfunctioning—are likely to be multiple, so that if only one is removed, there may well be another that is the cause of the persistent hypoglycemia. It is also true that sometimes the islet cell tumors do not arise from within the pancreas. We saw a functioning adenoma recently that was behind the spleen, entirely separate from the pancreas.

Case 16 (Hodgkin's Disease)

Diagnosis.—Hodgkin's disease with miliary tuberculosis.
Contributor.—Dr. Meissner.
History.—A 58 year old salesman was first admitted at age 52 because of chronic cough. Thoracotomy with biopsy of enlarged mediastinal lymph nodes showed "lymphocytoma." Postoperatively he received x-irradiation to the mediastinum and over the next few years several courses of irradiation to the neck and supraclavicular fossae. He carried on with fairly normal activity until 2 weeks before final admission, at which time he deteriorated rapidly with a daily fever of 102 F. to 104 F. and developed icterus, meningismus, and a white blood count of 2,200.

DR. MEISSNER: In reviewing this case, we can start with the original biopsy taken from the mediastinal node which shows a diffuse replacement of architecture by small cells resembling lymphocytes. The diagnosis on this was lymphocytoma or small cell type of lymphosarcoma. It is interesting that repeated irradiation treatment produced good palliation, and the patient continued to carry out normal

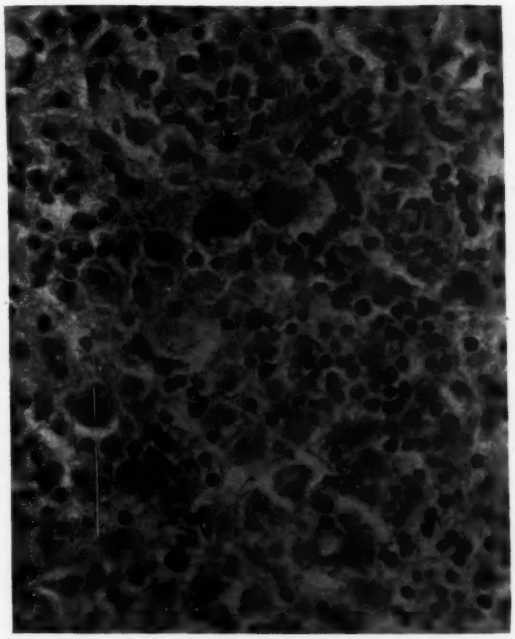

Fig. 23. Case 16. Hodgkin's disease with miliary tuberculosis. The lymphoid architecture has been replaced by pleomorphic cells of the lymphoid series and multinucleated cells of the Reed-Sternberg type are present.

activities until shortly before his death when he ran a rapid downhill course.

The tissue submitted to the seminar represents a lymph node found at autopsy and is typical of many of the lymph nodes from various parts of the body. The lymph node architecture is replaced by numerous foci of necrosis and a cellular infiltration of pleomorphic cells of the lymphoid series plus multinucleated giant cells with bean shaped nuclei. Some of the latter cells are typical of the Reed-Sternberg type.

There were other interesting autopsy findings, but before we talk of these it might be well to see the tabulated diagnoses, inasmuch as the lymph node was the only material available for the seminar.

Submitted Diagnoses.—Hodgkin's disease (with radiation change, 4) 14; lymphoma (radiation change, 5; necrosis, 1; reticulum cell, 1; giant follicular, 1) 8; radiation necrosis, 7; myeloid metaplasia (histoplasmosis?, 1) 4; leukemia (aleukemic, 1; myelogenous, 1) 2; necrotizing granulomatosis, 2; histoplasmosis, 2; polycythemia vera, 1.

DR. MEISSNER: At autopsy there was malignant lymphoma which we classified as Hodgkin's disease, moderately widespread, of lymph nodes in many parts of the body, but also involving spleen, bone marrow, and right lung. The change of the type of lymphoma from a lymphocytoma to Hodgkin's disease is always of interest, and such fluidity in histologic pattern has been emphasized by Custer.[1] It is interesting that the process was controlled so well by x-ray therapy in spite of the fact that in some areas, at least, the tumor appears as a Hodgkin's sarcoma. So perhaps we can assume a recent change to a more malignant process.

The Hodgkin's disease, however, was not the primary cause of death in this patient. There was, in addition, a fibrocaseous pulmonary tuberculosis of the right upper lobe with miliary tuberculosis involving both lungs, spleen, liver, adrenal, kidneys, bladder, prostate, seminal vesicles, lymph nodes, thyroid, parathyroid, and bone marrow. The necrosis found in the lymph nodes was not as we initially had thought—due to Hodgkin's disease—but was due to the miliary tuberculosis. Acid-fast organisms were so abundant that tissues from this case were used as controls. The miliary tuberculosis, of course, explains the rapid downhill course of the patient during the last few weeks of his life, but the diagnosis was not made until after microscopic study of the autopsy tissues.

The association of lymphoma, particularly Hodgkin's disease, and tuberculosis has been noted many times but was perhaps appreciated better by some of the older pathologists than it is today. Ewing[3] in 1940 wrote that acute miliary tuberculosis often terminates Hodgkin's disease and quoted Ziegler as saying that 20 per cent of the cases show tuberculous lesions. It is a complication which must continually be suspected in a patient with lymphoma who has a sudden progressive deterioration.

Fig. 24. Case 16. There are numerous foci of necrosis.

DR. C. P. SCHWINN, Houston: I actually submitted a little late the diagnosis here of nonreactive tuberculosis in lymphoma. I learned about this from a resident who had a considerable interest in tuberculosis both experimentally and otherwise. There also have been a number of recent reports on this, especially in patients who had received cortisone.

DR. MEISSNER: This patient did not receive cortisone as far as I know.

DR. ROBERT HAUSMAN, San Antonio: I don't think the diagnosis of Hodgkin's disease has been established here. There are no prominent nucleoli in the giant cells.

DR. MEISSNER: I think some of the cells had prominent nucleoli. Perhaps they didn't show well on the screen, but we felt that some of them at least were Dorothy Reed cells without much question. I wouldn't quibble too much, however, if one wants to call this a reticulum cell sarcoma.

DR. J. B. HUTCHESON, Dallas: I belong in this minority that was voting myeloid metaplasia in this case, and thought I could demonstrate large numbers of nucleated red blood cells. Obviously you didn't consider this, but if you had, would you have considered a PAS stain of value in differentiating a megakaryocyte from a Reed-Sternberg cell?

DR. MEISSNER: We have tried that in our laboratory, and it hasn't been very helpful to us.

DR. SEVERANCE: In view of so many votes for histoplasmosis on the screen, were special stains done to demonstrate these organisms, or were you happy when you found acid-fast bacilli?

DR. MEISSNER: When we found acid-fast organisms we quit while we were ahead.

DR. SEVERANCE: Some time ago we saw a case which we interpreted as showing a lymphocytic type of lymphoma associated with what we thought was Hodgkin's disease. Do you see that very often?

DR. MEISSNER: I don't know how often one sees lymphocytic type of lymphoma turn into Hodgkin's disease, but we've certainly been impressed with what Custer calls the fluidity of these lesions, changing back and forth. It seems to me that Hodgkin's disease doesn't often change back to something else, but other lymphomas often change into Hodgkin's disease.

DR. SEVERANCE: What I meant was that we found the two together, lymphosarcoma in one specimen and Hodgkin's disease in another, both removed at the same time from a single patient.

DR. MEISSNER: I agree that is unusual.

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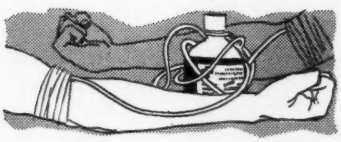
Dr. Meissner, New England Deaconess Hospital, Boston.
Lt. Col. Hansen, Chief, Anatomical Pathology Section, Brooke General Hospital, Brooke Army Medical Center, Fort Sam Houston.



CASE REPORTS

Malignant Carcinoid

Report of a Case Confined by Percutaneous Hepatic Biopsy

A case is reported of classical Carcinoid Syndrome in which the diagnosis was confirmed by elevated 5-hydroxyindole acetic acid in the urine and demonstration of carcinoid metastases in a percutaneous hepatic biopsy.

ROBERT H. MOSER, MAJOR, M.C., and
O. ROGER HOLLAN, M.D.

Fort Sam Houston, Texas

ONE OF THE more intriguing aspects of medicine is the discovery of a new syndrome. Quite often a combination of clinical findings will escape correlation until some alert physician encounters several baffling cases that present strikingly similar characteristics. After a period of reflective deduction and wandering through the literature, he may discover a denominator common to all of his cases. And so, the medical world is presented with a new syndrome.

Soon after initial publication, a swarm of reports suddenly materializes as everyone recalls a case that has been lying unsolved and fallow, gathering dust in his archives of clinical enigmas. Such was the evolution of malignant carcinoid which was first delineated as a syndrome by Millman[19] in 1943 and then elaborated and popularized by Biorck, Axen, and Thorson[1] in 1952.

The following patient was seen in consultation. She demonstrated many of the classical signs and symptoms of malignant carcinoid. Diagnosis was suspected on clinical evidence and confirmed by urinary 5-HIAA and percutaneous hepatic biopsy.

Case History

This is a 52 year old white housewife admitted to a local hospital in February, 1958. Her presenting complaints were diarrhea and daily temperature elevations (101 to 103 degrees) for the preceding 2 months. Her stools had

been quite loose and watery, numbering two to six each day, and occurring without periodicity day or night. There was ill defined "soreness" in the right hypochondrium, anorexia, and a 12 pound loss of weight in the 6 months prior to admission. Both abdominal pain and fever were relieved by ASA with predictable consistency.

Past medical history revealed that episodic attacks of fever and right upper quadrant pain had begun in 1953. At this time "genitourinary tract studies" had been negative. No hospital records were available for study. In 1955, hepatic enlargement had been reported on an abdominal radiograph. She also was noted to have had a sinus tachycardia of 100 to 130 at that time.

Admission physical examination revealed a pale, chronically ill-appearing, but pleasant middle-aged lady. She was neither dyspneic nor cyanotic. Blood pressure was 120/70, pulse rate 128, temperature 101 degrees (oral). The hepatic edge was palpable at 10 cm. below the right costal margin; it was smooth and nontender. No ill effects were noted after rather deep and vigorous palpation of her abdomen. The remainder of the physical examination was unremarkable.

Hemoglobin was 12.0 Gm., hematocrit 38 per cent, white blood count 8,600 with a normal differential distribution. Urinalysis, including sediment study, was negative. Total protein was 7.2 Gm. per 100 cc. with globulin of 3.2 and albumin of 4.0. Alkaline phosphatase was 12.8 BU. A qualitative test of 5-hydroxyindole acetic acid (5-HIAA) was positive. Stool examination was negative for ova and parasites. Complete upper and lower gastrointestinal examination was negative. Percutaneous punch biopsy of the liver was performed. Microscopic sections revealed clumps of tumor cells compatible with a diagnosis of carcinoid (see figures 1 and 2).

On the eighth hospital day abdominal exploration was undertaken. Small, pearly grey tumor nodules were found studding the peritoneal cavity. A small primary tumor, 1.0 cm. in diameter, was discovered lying in the subserosa of the proximal ileum. The appendix and right ovary were studded with tumor implants and were removed (see figure 3). The postoperative course was uneventful and the patient was discharged from the hospital in mid-March 1958.

Her temperature descended by lysis from the day of surgery until it reached normal by the sixth day. In subsequent weeks, she gained 2 pounds. However, her pulse rate persisted at 120 per minute, and she continued to experience occasional pain in the right hypochondrium. Otherwise she was devoid of symptoms. Several 24 hour urine collections were made in May, 1958, and sent to the National Institutes of Health (laboratory of Dr. Albert Sjoerdsma). These were reported to contain 29 and 33 mg. of 5-hydroxyindole acetic acid per 24 hour urine collection.

Comment

Some degree of optimism may be held for this patient since substantial clinical improvement has occurred since resection of the primary tumor and the metastatic masses involving the appendix and right ovary. With the disappearance of fever, diarrhea, and severe attacks of abdominal pain, we may hope that regression of distant metastases may occur. However, the persistent elevation of 5-HIAA in May indicates the continued presence of functioning carcinoid, probably in the liver. Survivals of 75 months[9] to 20 years[5] have been reported in this situation.

Discussion

Carcinoid tumor has been appreciated as a pathological entity for a long time. It is estimated to constitute about 0.4 per cent of all gastrointestinal tumors. Unsuspected carcinoids of the appendix occasionally are discovered in patients being operated upon for appendicitis or cholecystitis. Appendiceal carcinoid has enjoyed the reputation of being of low metastatic tendency. In MacDonald's comprehensive review of 356 carcinoids, 58 per cent were appendiceal and none of these had metastasized beyond the regional lymph nodes.[20] None had manifested "Carcinoid Syndrome." It is the primary carcinoid lying outside the appendix which has the metastatic propensity.[4]

Of 149 extraappendiceal carcinoids, MacDonald found a 16 per cent incidence of metastasis to lungs and/or liver. In other series this varies from 25 per cent[21] to 43 per cent.[26] The most common sites of metastases after regional nodes are retroperitoneal nodes, liver, lungs, spleen, pancreas, adrenals, kidneys, brain, marrow, and ovaries. Carcinoids have been identified as arising from the Kultschitzky cells of the chromaffin system lying at the base of the crypts of Lieberkuhn. Since this argentaffin cell has extensive distribution throughout the gastrointestinal tract, it is not surprising that carcinoid tumors have been located from stomach[29] to rectum.[14, 18, 24] Aside from the appendix, the ileum[9, 20] is the most frequent site of carcinoid formation. However, Carcinoid Syndrome has been reported in the presence of metastasizing pulmonary adenoma,[28] and in a nonmetastasizing dermoid cyst.[10]

Carcinoid Syndrome is quite rare as evidenced by 4 cases exhibiting the classic signs and symptoms out of 24 metastatic carcinoids from a total series of 356. This means that only about .01 per cent of all carcinoids exhibit the syndrome. However, familiarity with the characteristic symptom complex is justified since this knowledge facilitates the recognition of this rare entity to a degree of accuracy rarely attained in the area of tumor diagnosis. The recollection of any syndrome is a function of comprehending the genesis of its signs and symptoms. In carcinoid, the hallmark is production of excessive amounts of serotonin with its diverse and strange effects.

Serotonin is a provocative substance which has stimulated considerable pharmacological and clinical interest.[12, 13, 15, 22, 23] It has been called a hormone and is known to be a normal constituent of platelets, brain, and small intestine. It is felt that the amino acid tryptophan is first converted to 5-hydroxy-

tryptophan. A specific decarboxylase then converts this intermediary product into active 5-hydroxytryptamine which is serotonin. In patients with significant serotonin-producing carcinoids, a deficiency of tryptophan has been demonstrated.[23]

Serotonin in turn is oxidized by mono-amine-oxidase to 5-hydroxyindole acetic acid (5-HIAA), an excretory form which is detected readily in the urine by chemical means.

Let us now attempt to correlate the apparently diverse signs and symptoms of Carcinoid Syndrome under the common denominator of serotonin excess. The normal subject carries levels of 0.26 to 0.63 micrograms of serotonin/mg. of platelet protein. The patient with Carcinoid Syndrome may carry 1.0 to 5.6 micrograms/mg. of platelet protein. So there may be minimal overlapping. Apparently the effects of serotonin excess are related to a specific, selective ability to constrict smooth muscle in addition to other less well defined effects. These may best be summarized by the following outline:

A. Smooth Muscle Spasm (selective).—1. This usually involves the arterioles of the face, neck, and upper chest, manifesting itself with a striking red to cyanotic papular rash ("carcinoid flush") which occurs episodically. Phases have been described in which there is an initial flush followed by cyanosis. It has been related to meals.[21] This mechanism would seem to be a combination of localized arteriolar spasm with capillary dilatation and stasis in the deeper skin layers. It may eventually terminate in telangiectasis formation if attacks continue to recur over a sufficiently prolonged period. A "pellagrous dermatitis" which responded to nicotinic acid[3] has been reported in a case of Carcinoid Syndrome.

2. It also is postulated that pulmonary arterioles may be involved. Most serotonin probably exists in an inert state bound to platelets. Sudden platelet disruption, as would occur with embolism and/or thrombus formation, may cause an acute release of serotonin. This is cited as a possible contributory mechanism in the acute cor pulmonale associated with pulmonary embolism. Frequently, the amount of cardiopulmonary disability is disproportionate to mechanical effects that would be predicted from the size of the embolism and retrograde thrombosis. Some other mechanism must be invoked. It may be that the sudden platelet disruption and ensuent release of platelet-bound serotonin to the free form causes significant arteriolar spasm. This mechanism has never been proven to complete satisfaction. Intravenous injection of serotonin into the pulmonary arteries of cats has produced pulmonary artery vasoconstriction and pulmonary embolism-like signs and symptoms.[8] In man, 5-hydroxytryptamine injection has caused elevation of PA and RV pressures.[23]

3. The smooth musculature of bronchioles may be involved, with signs of wheezing and symptoms of

Fig. 1. Low power view of hepatic biopsy revealing two distinct "nests" of argentaffine (carcinoid cells) in the midst of normal parenchyma.

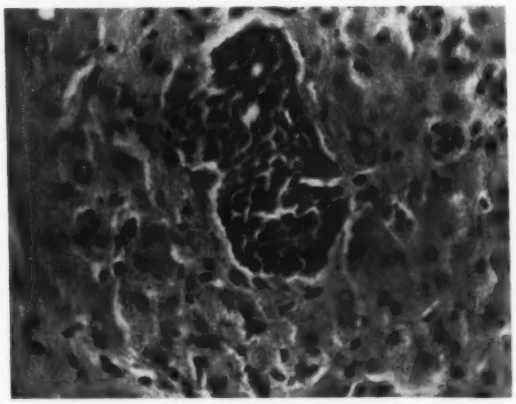


Fig. 2. High power view of "nest" of argentaffine (carcinoid) cells revealing typical configuration.

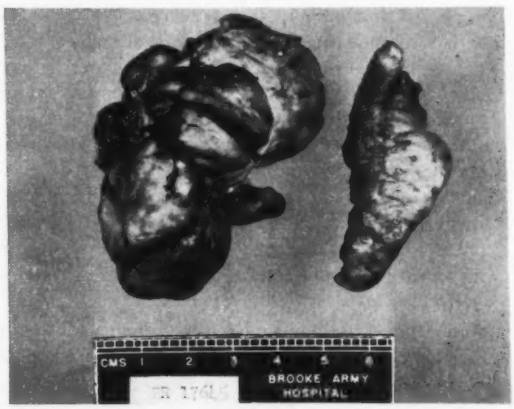


Fig. 3. Resected specimen from patient revealing primary tumor lying in the subserosa of the terminal ileum with tumor metastases in adjacent nodes and appendix. The extensively involved ovary is to the right.

dyspnea. The smooth musculature of the intestine may be provoked into spasm with ensuing diarrhea, flatulence, nausea, and vomiting. Even the lacrimal glands may be stimulated to induce excessive lacrimation and periorbital edema.

B. Hepatomegaly.—Hepatomegaly may be present with the liver edge smooth as the result of metastasis or right-sided cardiac failure. Rarely, it may be somewhat nodular from metastases involving the surface of the liver. Masses in the right lower quadrant are rare, but should be sought and could represent the initial tumor or localized lymph node involvement.

C. Cardiopathy Restriction to Right Ventricle.—By far the most fascinating aspect of Carcinoid Syndrome is the cardiopathy restricted to the right ventricle. This may be quite variable. It has manifested itself as marked pulmonary valvular stenosis with outflow tract endocardial fibrosis and/or tricuspid stenosis and/or tricuspid incompetence. In other patients, it has been manifest as hemodynamically insignificant, minimal subendocardial sclerosis. The etiology and pathogenesis remain obscure, and consequently, the door is left ajar for vigorous intellectual gymnastics.

It does not require great imagination to envision the poisonous serotonin-rich effluvia from hepatic metastases pouring into the inferior vena cava and "streamlining" into the ventricle to spare the right atrium. From here it could pass into the pulmonary system which is rich in amine oxidase. This enzyme is capable of oxidizing 5-hydroxytryptamine into 5-hydroxyindole acetic acid. It is this innocuous substance which is then delivered to the left heart.

Goble and his associates catheterized one patient with Carcinoid Syndrome and demonstrated a level of plasma serotonin in the right ventricle which exceeded that of the brachial artery threefold.[16] Attempts to confirm this phenomenon in other patients have been unrewarding.[23] Five-hydroxytryptamine (serotonin) in plasma exists in an active free state and inactive platelet-bound state. The plasma determination of serotonin is technically difficult. Sources of error are subtle, since platelets traumatized during separation of plasma from formed elements contribute falsely elevated levels of free serotonin. Conversely, if free serotonin is adsorbed to platelets falsely low levels may be reported.

The actual mechanism of serotonin assault upon the endocardium remains a source for speculation. A "direct chemical irritation" action has been proposed,[23] but this is difficult to visualize in a structure that is as relatively hardy as the endocardium. Since the inner few millimeters of endocardium derive nutrition by oxygen diffusion from intracavitary blood, a mechanism premised on coronary insufficiency seems unlikely. Another possibility suggested by Thorson[23] is that altered cardiac dynamics during flush episodes may damage the heart. Apparently, at the onset of a flush, stroke volume and minute output are decreased. During the peak of the flush, output increases and the heart may dilate only to have output decrease during the final phase of the flush. It is postulated that this may cause "stretch" damage to valves. However, this would not explain the localization to the right heart.

An interesting corollary is the suggestion of Waldenstrom[27] that the entity, subendocardial fibroelastosis, may be caused by an excess of serotonin in the fetal circulation. It would be somewhat difficult to explain the predominance of left ventricular endocardial damage which characterizes this process since all portions of the fetal heart should be bathed by the serotonin. Perhaps "streamlining" from the inferior vena cava through the foramen ovale, sparing the right ventricle, may be an acceptable explanation.

Clinically, the patient with Carcinoid Syndrome may demonstrate classic signs of pulmonary stenosis. These include precordial heave, normal to soft pulmonic second sound, harsh basal systolic murmur, and possibly signs of right heart failure. Electrocardiography may indicate hypertrophy of the free wall of the right ventricle with a dominant right precordial vector. A low-pitched diastolic rumble may represent tricuspid stenosis. Often the involvement may be combined tricuspid and pulmonary valvular stenosis, quite analogous to the rheumatic fever effects on the left-sided valves.

D. Carcinoid Syndrome.—Cerebral symptoms have been reported and may be related to a "pellagra" like disturbance related to a nicotinamide deficiency since both 5-OH tryptamine and Vitamin B compete for the tryptophan substrate.[6] This is related to the "pellagrous" skin changes (vide supra).

It may be well to spend a few moments on the rarity of the syndrome in contrast to the number of proven cases of carcinoid. You will recall the .01 per cent incidence of Carcinoid Syndrome in cases of histologically proven carcinoid tumor, which of themselves constitute 0.4 per cent of all gastrointestinal tumors. It would be apparent that certain distinct conditions must exist for the tumor to produce the syndrome.

The chief requirement would appear to be hepatic metastases.[22] It is not certain that all carcinoids have the capability of producing serotonin,[23] and apparently the liver is able to detoxify all serotonin that is produced within the portal drainage area. One may speculate as to whether a primary gastrointestinal carcinoid could produce vast quantities of serotonin,

sufficient to overtax the mono-amine oxidase system of the liver with the escape of enough serotonin to invoke the syndrome. Dockerty has stated ". . . all one requires for production of the Carcinoid Syndrome is a large mass of carcinoid tissue, located anywhere in the body. Nonmetastasizing carcinoids do not elicit the syndrome because they are too small."[10]

Documented Carcinoid Syndrome has been observed in 2 patients who had pulmonary adenomas with hepatic metastasis.[28] Again, one may wonder if such a serotonin-producing tumor without metastasis could discharge its hormone into the pulmonary or bronchial veins to by-pass and/or overwhelm both pulmonary and hepatic "detoxification stations" and cause the syndrome. Suffice to say that with one notable exception (a case of dermoid cyst containing carcinoid implants without metastasis),[10] all other instances of Carcinoid Syndrome have occurred in the presence of hepatic metastases.

Diagnosis

Diagnosis of carcinoid can be confirmed by the demonstration of 5-HIAA in the urine. There is no other disease or syndrome known that is capable of significantly elevating 5-HIAA. Low values have been found in various collagen diseases.[17] It is rare to have a definitive laboratory test to confirm a clinical diagnosis without equivocation or qualification. Normal urinary levels of 5-HIAA range from 2-9 mgm./day. In proven cases of Carcinoid Syndrome it varies from 200 to 900 mg. per day.[23] This is a chemical determination that can be accomplished without too much difficulty. The only requirement is to insure careful collection of several 24 hour urine specimens.

It must be recalled that the catechol amines of pheochromocytoma demonstrate a crucial capriciousness and ideally should be collected during an attack or following histamine provocation. In contrast, the carcinoid is much more stable and produces serotonin more constantly. However, Sjoerdsma and others[23] have found higher levels of 5-HIAA during flush episodes although plasma serotonin (free or platelet-bound) remained unchanged. These authors feel that 5-HIAA is a "sensitive index of the mass of functioning tumor" and suggest that its measurement may be a useful tool in following the course of the disease and in evaluating therapy designed to eradicate the tumor or prevent formation of serotonin.[19]

Major Robert H. Moser was formerly assistant chief of the Department of Medicine at Brooke Army Medical Center, Fort Sam Houston, and is at present attached to the Department of Medicine, University of Utah College of Medicine.

Therapy

Treatment of carcinoid syndrome may be divided into surgical and medical aspects. Carcinoids in any location should be resected if at all possible. Dockerty has stated that it may take up to 25 years (at the extreme) for a carcinoid to metastasize and impair vital organs.[11] Survivals of over 75 months[9] to 20 years[5] are reported following resection of primary sites in the presence of metastases. Regression of metastases after resection of the primary site also has been observed. Additional evidence to recommend resection can be found in the tendency of carcinoid and its local metastases to induce mechanical obstruction of the gastrointestinal tract.[2, 9, 29]

Drug therapy in carcinoid is perplexing. It is fascinating in theory but valueless in practice. In animals Rauwolfia derivatives, notably large doses of Serpasil (3-5 mgm./kg.) tend to drive serotonin from cerebral, platelet, and small intestinal depots with a measurable elevation of serotonin in plasma and of 5-HIAA in the urine.[17] Haverback et al found that doses of Reserpine up to 30 micrograms/kg. in man lowered serotonin levels in platelets but not in the gastrointestinal tract. He was unable to detect elevation of 5-HIAA and attributed it to the fact that the gastrointestinal tract is the largest serotonin depot and quantitative measurements of 5-HIAA "may not detect alterations of serotonin unless this depot is affected or there is a marked overproduction . . . by a tumor."[17] Warner and Southren report a significant relief of carcinoid symptoms in one of their patients with pulmonary adenoma, and demonstrated recrudescence upon cessation of Raudixin therapy.[28]

A curious syndrome has been ascribed to Serpasil in which the induced depletion of brain serotonin disturbs the physiologic antagonism between serotonin and the endogenous hallucinogen, lysergic acid diethylamide. This results in unantagonized lysergic acid diethylamide with some rather untranquil hallucinations indirectly precipitated by this "tranquilizing" agent. Exogenous lysergic acid diethylamide has been observed to produce symptoms of serotonin excess in two patients, a perverse effect which eludes current explanation. An analogue of lysergic acid diethylamide (2 brom, D-lysergic acid) has been reported to antagonize the smooth muscle effects of serotonin. This has not been demonstrated with any consistency in other laboratories. Thus, one can see the complexity of the interrelationship of these substances which awaits eludication.

Chlorpromazine, the ubiquitous antiemetic, hibernator, tranquilizer, diuretic, occasional icterus-inducer, and marrow depressor, also has been reported to diminish 5-HIAA excretion[7] and afford symptomatic relief in Carcinoid Syndrome.[23] Promazine has been related to relief of symptoms in one case.[11]

Conclusions

We have reported another case of classical Carcinoid Syndrome in which the diagnosis was confirmed by elevated 5-HIAA in the urine and a demonstration of carcinoid metastases in a percutaneous hepatic biopsy. The opinion is expressed that familiarization with the clinical phenomena of the syndrome is justifiable since the diagnosis can be strongly suspected on clinical grounds and readily confirmed by assay of the urine for 5-HIAA.

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Dr. Moser, Department of Medicine, University of Utah College of Medicine, Salt Lake City.

Dr. Hollan, Brooke Army Hospital, Brooke Army Medical Center, Fort Sam Houston.

MEDICAL NEWS

★ Coming Meetings

Texas Medical Association, Austin, Sept. 17-18, 1960, Jan. 28, 1961; Galveston, April 23-25, 1961. Dr. May Owen, Fort Worth, Pres.; Mr. C. Lincoln Williston, 1801 North Lamar Blvd., Austin, Exec. Secy.

American Medical Association, Washington, D. C., Nov. 28-Dec. 1, 1960. Dr. Louis M. Orr, Orlando, Fla., Pres.; Dr. F. J. L. Blasingame, 535 North Dearborn, Chicago 10, Exec. Vice-Pres.

Current Meetings

JULY

Eighth District Society, Corpus Christi, July 8-9, 1960. Dr. M. Warren Hardwick, Angleton, Pres.; Dr. J. L. Coleman, Box 3346, Victoria, Secy.

Postgraduate Medical Assembly of South Texas, Houston, July 18-20, 1960. Dr. C. Alsworth Calhoun, Houston, Pres.; Mrs. W. H. Dahme, 412 Jesse H. Jones Library Bldg., Houston 25, Exec. Secy.

AUGUST

American Congress of Physical Medicine and Rehabilitation, Washington, D.C., Aug. 21-26, 1960. Dr. F. J. Kottke, Minneapolis, Pres.; Dorothea C. Augustin, 30 N. Michigan Ave., Chicago 2, Executive Secy.

American Hospital Association, San Francisco, Aug. 29-Sept. 1, 1960. Dr. Russell A. Nelson, Baltimore, Md., Pres.; Dr. Edwin L. Crosby, 18 E. Division Street, Chicago, Executive Director.

National and Regional

American Academy of Allergy, Washington, D. C., Feb. 6-8, 1961. Dr. Bram Rose, Montreal, Canada, Pres.; Dr. Joseph Noah, 100 N. Euclid Ave., St. Louis 8, Mo., Secy.

American Academy of General Practice, Miami, April 17-20, 1961. Dr. John G. Walsh, Sacramento, Calif., Pres.; Mr. Mac F. Cahal, Volker Blvd. at Brookside, Kansas City 12, Executive Secy.

American Academy of Pediatrics. Dr. William W. Belford, San Diego, Calif., Pres.; Dr. E. H. Christopherson, 1801 Hinman Ave., Evanston, Ill., Executive Director.

American Association for Thoracic Surgery. Dr. William E. Adams, Chicago, Pres.; Dr. Hiram T. Langston, 7730 Corondelet Ave., St. Louis 5, Secy.

American Association of Genito-Urinary Surgeons. Dr. Reed M. Nesbitt, Ann Arbor, Mich., Pres.; Dr. W. J. Engel, 2020 E. 93rd St., Cleveland 6, Secy.

American Association of Plastic Surgeons. Dr. Lyndon A. Peer, New Jersey, Pres.; Dr. Thomas D. Cronin, 6615 Travis St., Houston 25, Secy.

American College of Allergists, Dallas, March 12-17, 1961. Dr. Giles A. Koelsche, Rochester, Minn., Pres.; Mr. Eloi Bauers, 2160 Rand Tower, Minneapolis, Executive Vice-Pres.

American Academy of Dermatology and Syphilology, Chicago, Dec. 3-8, 1960. Dr. Francis W. Lynch, St. Paul, Minn., Pres.; Dr. Robert R. Kierland, Mayo Clinic, Rochester, Minn., Secy.

American Academy of Ophthalmology and Otolaryngology, Chicago, Oct. 9-14, 1960. Dr. John H. Dunnington, New York, Pres.; Dr. W. L. Benedict, 15 Second St. S.W., Rochester, Minn., Secy.

American Association for Maternal and Infant Health. Dr. M. Edward Davis, Chicago, Pres.; Mrs. Patricia Dorr, 116 S. Michigan Ave., Chicago 3, Executive Director.

American Association of Obstetricians and Gynecologists, Hot Springs, Va., Sept. 8-10, 1960. Dr. Robert A. Ross, Chapel Hill, N. C., Pres.; Dr. Clyde L. Randall, 216 Summer St., Buffalo 22, Secy.

American Cancer Society, New York, Oct. 26-27, 1960. Dr. Warren H. Cole, Chicago, Pres.; Mr. Granville Whittlesey, 521 West 57th St., New York 19, Secy.

American College of Chest Physicians. Dr. Seymour M. Farber, San Francisco, Pres.; Mr. Murray Kornfeld, 112 E. Chestnut, Chicago 11, Executive Director.

American College of Gastroenterology, Philadelphia, Oct. 23-29, 1960. Dr. Joseph Shaiken, Milwaukee, Pres.; Mr. Daniel Weiss, 33 West 60th, New York 23, Executive Director.

American College of Obstetricians and Gynecologists. Dr. John I. Brewer, Chicago, Pres.; Mr. Donald F. Richardson, P. O. Box 749, Chicago 90, Executive Secy.

American College of Physicians. Dr. Howard P. Lewis, Portland, Ore., Pres.; Mr. E. R. Loveland, 4200 Pine, Philadelphia 4, Secy.

American College of Radiology, Atlantic City, Sept. 30-Oct. 1, 1960. Dr. Earl E. Barth, Chicago, Pres.; Mr. W. C. Stronach, 20 N. Wacker Dr., Chicago 6, Executive Director.

American College of Surgeons, San Francisco, Oct. 10-14, 1960. Dr. Owen H. Wangensteen, Minneapolis, Pres.; Dr. William E. Adams, 950 E. 59th St., Chicago, Secy.

American Dermatological Association. Dr. Marion B. Sulzberger, New York, Pres.; Dr. Wiley M. Sams, 308 Ingraham Bldg., Miami 32, Secy.

American Gastroenterological Association. Dr. H. Marvin Pollard, Ann Arbor, Pres.; Dr. Wade Volwiler, University of Washington School of Medicine, Seattle 5, Secy.

American Gynecological Society. Dr. Karl H. Martzloff, Portland, Pres.; Dr. A. A. Marchetti, 3800 Reservoir Rd. N.W., Washington 7, D.C., Secy.

American Heart Association, St. Louis, Oct. 21-25, 1961. Dr. A. Carlton Ernstene, Cleveland, Pres.; Mr. William F. McGlone, 44 E. 23rd, New York 10, Secy.

American Laryngological, Rhinological, and Otological Society, Lake Placid Club, May 23-25, 1960. Dr. Fletcher D. Woodward, Charlottesville, Va., Pres.; Dr. C. S. Nash, 708 Medical Arts Bldg., Rochester, N. Y., Secy.

American Neurological Association. Dr. Derek Denny-Brown, Boston, Pres.; Dr. Melvin D. Yahr, New York Neurological Institute, 710 W. 168th Street, New York 32, Secy.

American Ophthalmological Society. Dr. A. B. Reese, New York, Pres.; Dr. M. C. Wheeler, 30 W. 59th, New York 19, Secy.

American Orthopaedic Association. Dr. John Royal Moore, Philadelphia, Pres.; Dr. Lee Ramsay Straub, 535 East 70th St., New York 21, Secy.

American Pediatric Society. Dr. Samuel Z. Levine, New York, Pres.; Dr. A. C. McGuinness, Room 1036, 2800 Quebec St., N.W., Washington 8, D.C., Secy.

American Proctologic Society. Dr. H. R. Reichman, Salt Lake City, Pres.; Dr. Norman D. Nigro, 10 Peterboro, Detroit 1, Secy.

American Psychiatric Association. Dr. William Malamud, New York, Pres.; Dr. C. H. Hardin Branch, 156 Westminister Ave., Salt Lake City, Secy.

American Public Health Association, San Francisco, Oct. 31-Nov. 4, 1960. Dr. Malcolm H. Merrill, Berkeley, Calif., Pres.; Dr. Berwyn F. Mattison, 1790 Broadway, New York 19, Executive Secy.

American Society of Anesthesiologists, New York, Oct. 2-7, 1960. Dr. J. Earl Remlinger, Jr., Wilmette, Ill., Pres.; Dr. Robert L. Patterson, 612 Bershire Drive, Pittsburgh 15, Secy.

American Surgical Association, White Sulphur Springs, W. Va., April 3-6, 1960. Dr. Warren H. Cole, Chicago, Pres.; Dr. William Altemeier, Cincinnati General Hospital, Cincinnati 29, Secy.

American Society of Clinical Pathologists, Chicago, Sept. 24-Oct. 2, 1960. Dr. John J. Clemmer, Albany, Pres.; Mr. Claude E. Wells, 445 Lake Shore Drive, Chicago 11, Executive Secy.

American Urological Association. Dr. William M. Coppridge, Durham, N. C., Pres.; Mr. William P. Didusch, 1120 N. Charles St., Baltimore 1, Executive Secy.

Association of American Physicians and Surgeons, St. Louis, Sept. 29-Oct. 1, 1960. Dr. Louis Wegryn, Elizabeth, N. J., Pres.; Mr. Harry E. Northam, 185 N. Wabash Ave., Chicago 1, Executive Director.

International College of Surgeons, U. S. Chapter. Dr. Henry Meyerding, Rochester, Minn., Pres.; Dr. Ross T. McIntire, 1516 Lake Shore Dr., Chicago, Executive Director.

National Tuberculosis Association. Dr. H. McLeod Riggins, New York, Pres.; Mrs. Wallace B. White, 651 Marlborough Rd., Brooklyn, Secy.

Radiological Society of North America, Cincinnati, Ohio, Dec. 4-9, 1960. Dr. Lawrence L. Robbins, Boston, Pres.; Dr. Donald S. Childs, 713 E. Genesee, Syracuse 2, N. Y., Secy.

South Central Association of Blood Banks. Dr. John B. Alsever, Phoenix, Pres.; L. Ruth Guy, Ph.D., Room 1101, Stoneleigh Hotel, Dallas, Secy.

Southern Medical Association, St. Louis, Oct. 31-Nov. 3, 1960. Dr. Edwin H. Lawson, New Orleans, Pres.; Mr. Robert F. Butts, 2601 Highland Ave., Birmingham 5, Ala., Executive Secy.

Southern Psychiatric Association, Virginia Beach, Va., Oct. 2-4, 1960. Dr. David A. Wilson, Charlottesville, Va., Pres.; Dr. Richard Proctor, Winston-Salem, N. C., Secy.

Southern Surgical Association, Boca Raton, Fla., Dec. 6-8, 1960. Dr. Francis Massie, Pres.; Dr. John D. Martin, Jr., Emory University, Ga., Secy.

Southwest Allergy Forum. Dr. Johnny A. Blue, Oklahoma City, Pres.; Dr. George L. Winn, Suite 104, Lister Medical Building, 430 Northwest Twelfth St., Oklahoma City, Secy.

Southwest Regional Cancer Conference, Fort Worth, Oct. 23, 1960. Dr. Robb Rutledge, Fort Worth, Chm.; Mrs. Ira Frances Ball, Westchester House, Fort Worth, Secy.

Southwestern Medical Association, El Paso, Oct. 20-22, 1960. Dr. R. L. Deter, El Paso, Pres.; Dr. Merle Thomas, 1501 Arizona Building, El Paso, Secy.

Southwestern Society of Nuclear Medicine, San Antonio. Dr. Peter E. Russo, Oklahoma City, Pres.; Dr. J. R. Maxfield, Jr., 2711 Oak Lawn Avenue, Dallas, Secy.

Southwestern Surgical Congress, Fort Smith, Ark. Dr. Fred H. Krock, Fort Smith, Ark., Pres.; Mary O'Leary, 813 Medical Arts Bldg., Oklahoma City, Exec. Secy.

Tri-State Medical Assembly, Louisiana, Sept. 14-15, 1960. Dr. R. B. Langford, Shreveport, Pres.; Dr. J. W. Wilson, Jr., 940 Margaret Place, Shreveport, Secy.

United States-Mexico Border Public Health Association. Mr. Frank J. Von Zuben, Jr., Austin, Pres.; Dr. Jorge Román, 243 United States Court House, El Paso, Secy.

State

Private Clinics and Hospitals, Dallas, December, 1960. Dr. Vance Terrell, Stephenville, Pres.; Mr. B. J. Warren, Deaton Hospital, Galena Park, Secy.

Texas Academy of General Practice, Dallas, Oct. 3-5, 1960. Dr. E. Sinks McLarty, Galveston, Pres.; Mr. Donald C. Jackson, 1905 N. Lamar, Austin, Executive Secy.

Texas Academy of Internal Medicine. Dr. Merton M. Minter, San Antonio, Pres.; Dr. Hugo T. Engelhardt, 1216 Main, Houston, Secy. Meetings restricted to members.

Texas Air-Medics Association, Galveston, April 22-25, 1961. Dr. C. D. Henry, San Antonio, Pres.; Dr. C. F. Miller, Box 1338, Waco, Secy.

Texas Association for Mental Health, Galveston, 1961. Mr. William R. Ransone, Dallas, Pres.; Mrs. Lawrence Marcus, 3525 Arrowhead Drive, Dallas, Secy.

Texas Association of Obstetricians and Gynecologists, Houston, Feb. 10-11, 1961. Dr. Oran V. Prejean, Dallas, Pres.; Dr. Hugh W. Savage, 815 Fifth Ave., Fort Worth, Secy.

Texas Association of Public Health Physicians, Galveston, April 22-25, 1961. Dr. L. P. Walter, Austin, Pres.; Dr. W. V. Bradshaw, Jr., 1800 University Drive, Fort Worth, Secy.

Texas Club of Internists. Dr. W. W. Bondurant, Jr., San Antonio, Pres.; Dr. T. Haynes Harvill, Medical Arts Building, Dallas 1, Secy.

Texas Chapter, American Academy of Pediatrics, Galveston, April 22-25, 1961. Dr. J. T. Bennett, El Paso, Pres.; Dr. W. W. Kelton, Jr., 108 West 30th, Austin, Secy.

Texas Chapter, American College of Chest Physicians, Galveston, April 22-25, 1961. Dr. Hiram M. Anderson, San Angelo, Pres.; Dr. Milton V. Davis, 3707 Gaston Avenue, Dallas, Secy.-Treas.

Texas Dermatological Society, Galveston, April 22-25, 1961. Dr. M. W. Harrison, Houston, Pres.; Dr. D. Shelton Blair, 1609 Medical Arts Building, Dallas, Secy.-Treas.

Texas Diabetes Association, Galveston, April 22-25, 1961. Dr. James A. Greene, Houston, Pres.; Dr. John W. Chriss, 2436 Morgan Street, Corpus Christi, Secy.-Treas.

Texas Division, American Cancer Society. Dr. David A. Todd, San Antonio, Pres.; Mr. Curt W. Reimann, 5014 Bull Creek Rd., Austin 3, Executive Director.

Texas Heart Association, Dallas, Sept. 9-10, 1960. Dr. Robert E. Leslie, El Campo, Pres.; Mr. Ernest T. Guy, 404 Jesse H. Jones Library Building, Houston 25, Executive Director.

Texas Hospital Association. Mr. F. S. Walters, Jr., Amarillo, Pres.; Mr. O. Ray Hurst, 1905 N. Lamar, Austin, Executive Director.

Texas Industrial Medical Association, Galveston, April 22-25, 1961. Dr. Noble B. Daniel, Texarkana, Pres.; Dr. J. G. Burdick, Pasadena, Secy.

Texas Neuropsychiatric Association, Galveston, April 22-25, 1961. Dr. Clarence S. Hoekstra, Dallas, Pres.; Dr. E. Ivan Bruce, Jr., Galveston, Secy.-Treas.

Texas Ophthalmological Association, Galveston, April 22-25, 1961. Dr. Otto Lippmann, Austin, Pres.; Dr. James H. Scruggs, Waco, Secy.

Texas Orthopedic Association, Galveston, April 22-25, 1961. Dr. Ike S. McReynolds, Houston, Pres.; Dr. Margaret Watkins, Dallas, Secy.-Treas.

Texas Otolaryngological Association, Galveston, April 22-25, 1961. Dr. August J. Streit, Amarillo, Pres.; Dr. Louis E. Adin, Jr., Dallas, Secy.

Texas Pediatric Society, Sept. 30-Oct. 1, 1960, Dallas. Dr. Byron York, Houston, Pres.; Dr. C. E. Gilmore, 811 Bonham, Paris, Secy.

Texas Physical Medicine and Rehabilitation Society, Galveston, April 22-25, 1961. Dr. Edward M. Krusen, Dallas, Pres.; Dr. Oscar Selke, Medical Professional Building, Houston, Secy.

Texas Proctologic Society, Houston, February, 1961. Dr. J. Wade Harris, Houston, Pres.; Dr. H. Gray Carter, 915 St. Joseph St., Dallas, Secy.

Texas Public Health Association, Fort Worth, March 5-8, 1961. Mrs. Maggie Belle Davis, Corpus Christi, Pres.; Mr. Joseph N. Murphy, Jr., Box 4012, Austin 51, Executive Secy.

Texas Radiological Society, Fort Worth, Jan. 20-21, 1961. Dr. Delphin von Briesen, El Paso, Pres.; Dr. R. P. O'Bannon, 1216 Pennsylvania, Fort Worth, Secy.

Texas Rheumatism Association, Dallas, Dec. 9, 1960. Dr. Howard C. Coggeshall, Dallas, Pres.; Dr. J. Morris Horn, 3707 Gaston, Dallas, Secy.

Texas Society of Anesthesiologists, Galveston, April 22-25, 1961. Dr. Randle J. Brady, Houston, Pres.; Dr. Eugene L. Slataper, Houston, Secy.

Texas Society of Athletic Team Physicians, Galveston, April 22-25, 1961. Dr. W. S. Horn, Jr., Fort Worth, Pres.; Dr. Louis Levy, Fort Worth, Secy.-Treas.

Texas Society of Gastroenterologists and Proctologists, Galveston, April 22-25, 1961. Dr. H. Gray Carter, Dallas, Pres.; Dr. A. C. Broders, Jr., Temple, Secy.-Treas.

Texas Society of Pathologists, Inc., Galveston, April 22-25, 1961. Dr. O. J. Wollenman, Jr., Fort Worth, Pres.; Dr. Vernie A. Stembridge, Dallas, Secy.-Treas.

Texas Society of Plastic Surgeons, Galveston, April 22-25, 1961. Dr. J. B. Patterson, Fort Worth, Pres.; Dr. Raymond O. Brauer, Houston, Secy.-Treas.

Texas Society on Aging. Dr. Ernest W. Keil, Temple, Pres;. Mrs. William B. Ruggles, 3701 Stratford, Dallas, Secy.-Treas.

Texas State Board of Medical Examiners, Fort Worth, Dec. 1-3, 1960. Dr. M. H. Crabb, Fort Worth, Secy.; Mrs. Carolyn H. Millard, 1714 Medical Arts Bldg., Fort Worth, Assistant Secy.

Texas Surgical Society, San Antonio, Oct. 3-4, 1960. Dr. Robert Wilson Crosthwait, Waco, Pres.; Dr. G. V. Brindley, Jr., Scott and White Clinic, Temple, Secy.

Texas Society of Ophthalmology and Otolaryngology, San Antonio, Dec. 2-3, 1960. Dr. Lyle J. Logue, Houston, Pres.; Dr. Oliver Suehs, 14 Medical Arts Square, Austin, Secy.

Texas Traumatic Surgical Society, Galveston, April 22-25, 1961. Dr. Edward R. Rowe, Galveston, Pres.; Dr. John C. Long, Plainview, Secy.-Treas.

Texas Tuberculosis Association, Corpus Christi, March 16-18, 1961. Dr. J. Edward Johnson, Pres.; Miss Pansy Nichols, P. O. Box 6158, Austin 21, Executive Director.

Texas Urological Society, Wichita Falls, January 21-22, 1961. Dr. Joseph Mitchell, Dallas, Pres.; Dr. O. C. Berg, 1300 8th St., Wichita Falls, Secy.

District

First District Society. Dr. Russell Holt, El Paso, Pres.; Dr. Gordon L. Black, 1501 Arizona St., El Paso, Secy.

Second District Society, Snyder, 1961. Dr. M. J. Loring, Midland, Pres.; W. S. Parks, Jr., 2009 W. Wall, Midland, Secy.

Third District Society, Lubbock. Dr. Grady M. Wallace, Lubbock, Pres.; Dr. H. Fred Johnson, 2308 W. Eighth, Amarillo, Secy.

Fourth District Society. Dr. S. Braswell Locker, Brownwood, Pres.; Dr. J. G. Bodenhamer, Mason, Secy.

Fifth and Sixth Districts Society, Corpus Christi, July 8-9, 1960. Dr. John W. Chriss, Pres.; Dr. James Gabbard, 1001 Louisiana, Corpus Christi, Secy.

Seventh District Society. Dr. Robert N. Snider, Austin, Pres.; Dr. Richard Lucas, 502 W. 13th, Austin, Secy.

Ninth District Society, Bellville, Spring, 1961. Dr. Irving M. Watson, Jr., Conroe; Dr. William E. Sharp, 721 E. Texas, Baytown, Secy.

Tenth District Society, September, 1960. Dr. J. W. McCall, Jr., Beaumont, Pres.; Dr. Irving M. Richman, 3280 Fannin St., Beaumont, Secy.

Eleventh District Society. Dr. Marlin T. Braswell, Henderson, Pres.; Dr. Floyd Verheyden, 813 John St., Jacksonville, Secy.

Twelfth District Society, Waco, January 10, 1961. Dr. Bernard Rosen, Corsicana, Pres.; Dr. R. L. Campbell, 205 S. 15th St., Corsicana, Secy.

Thirteenth District Society, Fort Worth, Fall, 1960. Dr. William B. Allensworth, Mineral Wells, Pres.; Dr. R. D. Moreton, 1217 W. Cannon, Fort Worth, Secy.

Fifteenth District Society. Dr. Charles Wise, Naples, Pres.; Dr. George Bennett, 402 S. Bolivar, Marshall, Secy.

Clinics

Blackford Memorial Cancer Lectures, Denison, Nov. 11, 1960. Dr. Andrew Jackson, Denison, Chm.

Dallas Southern Clinical Society Conference, Dallas, March 20-22, 1961. Dr. Frank H. Kidd, Jr., Dallas, Pres.; Millard J. Heath, 433 Medical Arts Bldg., Dallas 1, Executive Officer.

International Medical Assembly of Southwest Texas, San Antonio, Jan. 23-25, 1961. Dr. Max E. Johnson, San Antonio, Pres.; S. E. Cockrell, 202 W. French Place, San Antonio, Exec. Secy.

New Orleans Graduate Medical Assembly, New Orleans, March 6-9, 1961. Dr. Maurice E. St. Martin, New Orleans, Pres.; Mrs. Irma B. Sherwood, 430 Tulane Ave., New Orleans 12, Executive Secy.

North Texas-Southern Oklahoma Fall Clinical Conference. Dr. Frank J. Lee, 1300 8th, Wichita Falls, Chm.

Oklahoma City Clinical Society Conference, Oklahoma City, Oct. 24-26, 1960. Dr. Vernon D. Cushing, Oklahoma City, Pres.; Miss Alma F. O'Donnell, 503 Medical Arts Bldg., Oklahoma City 2, Executive Secy.

Board Examinations

Texas State Board of Examiners in Basic Sciences, Austin, Houston, Galveston and Dallas. Henry B. Hardt, Ph.D., Fort Worth, Pres.; Mrs. Betty J. Anderson, Chief Clerk, State Office Bldg., 201 E. 14th St., Austin.

MEDICOLEGAL NOTES

Suits Sometimes Based On Departure from Procedures

Frequently, we have the question raised by physicians in Texas as to whether or not the omitting of certain tests or procedures ultimately might be used as a basis for establishing negligence in a malpractice case. An example might serve to demonstrate.

Let us take a situation where a physician feels that a preoperative test for the "bleeding-clotting" time of a patient is unnecessary, and consequently such test is omitted. The operation is performed but the result is unsatisfactory. Subsequently, the patient sues the physician for malpractice and alleges that the injury or harm suffered would not have happened had the physician performed the test for the "bleeding-clotting" time.

To better understand the result we might anticipate the courts ultimately will arrive at, it would be helpful to look to some of the basic principles which our courts long have adhered to in malpractice cases. First, our courts in Texas uniformly have taken the position that a physician is not a guarantor of a good or successful result in the care and treatment of his patients. So long as the physician has exercised in his care and treatment of the patient that degree of care and skill that would have been exercised by the average physician, in the same or a similar locality, under the same or similar conditions, the mere fact that an unfavorable result has been reached does not establish negligence on the part of the physician. To establish negligence on the part of the physician, the patient must establish that his physician in some way or manner failed or neglected to exercise that degree of skill or care that would have been exercised by the average physician in the same or a similar locality under the same or similar conditions.

In the example above, if it could be established that the average physician in the locality would not have omitted a preoperative test for the "bleeding-clotting" time of the patient, then liability may result if it can be shown that the omission of such test resulted in harm or injury to the patient. Consequently, when a physician is considering a departure from a course of care and treatment that is recognized and accepted widely, there is danger that such departure might be considered negligence on such physician's part unless it can be shown that this type of departure also is practiced by other physicians in the locality. It might be added that if such departure is fairly widely recognized throughout medicine as a whole, it certainly has the effect of lessening the possibility of any allegation that this departure was of a negligent nature.

Therefore, physicians should be cognizant that any significant departure from commonly recognized and accepted courses of care and treatment are not without legal implications and dangers unless it can be shown that the average physician in the locality would have followed a similar course of action under like circumstances.

—PHILIP R. OVERTON, LL.B., Austin.

MEDICAL MEETINGS

Texas Heart Association to Meet In Dallas, September 8-10

The Texas Heart Association will hold its 1960 annual meeting at the Adolphus Hotel in Dallas, September 8-10.

Subjects to be featured on the program are coronary artery disease, surgery and heart disease, and high blood pressure.

Among the out-of-state speakers will be Dr. A. Carlton Ernstene, Cleveland; Dr. Paul Oglesby, Chicago; Dr. S. Gilbert Blount, Jr., Denver; Dr. Robert P. Glover, Philadelphia; and Dr. Robert W. Wilkins, Boston.

Texans on the program will be Dr. Carleton B. Chapman, Dallas; Dr. Robert E. Leslie, El Campo; Dr. Marvin D. Siperstein, Dallas; Dr. James J. Leonard, Galveston; Dr. Raymond D. Pruitt, Houston; Dr. William S. Fields, Houston; Dr. Alfred W. Harris, Dallas; Dr. Hugh E. Wilson, Dallas; Dr. James Warren, Galveston; and Dr. Donald W. Seldin, Dallas.

The program will consist of some of the following topics: "What to Tell the Patient with Heart Disease," "The Ability of the Patient with Heart Disease to Perform Work," "A Long Term Study of Coronary Heart Disease," "The Clinical Evaluation of Operable Congenital Heart Disease and Indications for Surgery," "The Clinical Significance of Abnormal Heart Sounds," "The Secret of Success in the Surgery of Acquired Valvular Heart Disease," "Untreated Hypertension," "Neurological Disorders Related to Alterations in Blood Pressure," and "Medical Management of Essential Hypertension."

Additional information may be obtained by writing to the Texas Heart Association, 404 Jesse Jones Library Building, Texas Medical Center, Houston 25.

World Medical Association Assembles September 15

The German Medical Association will be host for the World Medical Association September 15-22 in Berlin.

The German Medical Association has scheduled its own annual meeting in conjunction with the fourteenth General Assembly of the world meeting. The opening and closing plenary sessions of the two organizations will be held together.

All activities will be conducted in the new Berlin Convention Hall, and the annual dinner on September 19 will be held at the "Palais am Funkturm." Featured on September 22 will be a sightseeing tour through the East Sector of Berlin and a cruise on Havel Lake.

Besides the regular features of the annual assembly, an international film program will spotlight postgraduate education.

Texas Hospital Group Holds Annual Meeting

Speakers at the Texas Hospital Association's thirty-first annual meeting May 10, 11, and 12, in Dallas included representatives of the medical profession, a United States congressman from Texas, and a national commentator.

Bruce Alger, United States congressman from Dallas, was guest speaker for a May 9 luncheon for the Association's House of Delegates, and Paul Harvey, president of Paul Harvey News, Chicago, was annual banquet speaker on May 12. His subject was "Two Worlds."

Dr. F. J. L. "Bing" Blasingame, executive manager of the American Medical Association, also was a guest speaker on May 10.

Other speakers and their topics: Frank S. Groner, president-elect of the American Hospital Association and administrator of the Baptist Memorial Hospital, Memphis, Tenn., "The Patient, End-Product or By-Product"; Richard K. Young, Th.D., chaplain of Baptist Hospital, Winston-Salem, N. C., and Dr. Milford O. Rouse, Dallas, "Spiritual Medicine, Treating the Patient as Well as the Disease"; Philip R. Overton, Texas Medical Association and Texas Hospital Association legal counsel, Austin, "From the Legal Angle—1959 Review and Forecast for 1960-1961."

Also, Lt. Col. Vincent I. Hack, M.S.C., Brooke Army Medical Center, Fort Sam Houston, "Color Dynamics in the Modern Hospital"; Ray E. Brown, "An Administrators' View of National Nursing Service"; Eleanor C. Lambertsen, secretary, Nursing Committee, American Hospital Association, Chicago, "Professional Nurses View."

Officers elected by the Association include Fred R. Higginbotham, Baptist Memorial Hospital, San Antonio, president; Albert H. Scheidt, Dallas County Hospital District, Dallas, president-elect; George B. Pearson, Medical Center Hospital, Tyler, vice-president; and Dr. Leigh J. Crozier, Houston, treasurer.

Next meeting of the association was set for May 14-18, 1961, in Dallas with headquarters in the Statler-Hilton Hotel.

Meeting in conjunction with the Hospital Association were the Texas Association of Hospital Auxiliaries, Texas Association of Medical Record Librarians, Texas Association of Nurse Anesthetists, Texas Association of Operating Room Nurses, Hospital Purchasing Agents Association of Texas, Association of Catholic Chaplains in Texas Hospitals, Hospital Chaplains in Texas, and Texas Conference of Catholic Hospitals.

Cancer Conference in Minneapolis

"Changing Concepts Concerning Cancer," is the theme of the fourth National Cancer Conference, to be sponsored by the American Cancer Society and the National Cancer Institute, September 13-15, in Minneapolis.

Among the presentations will be symposiums on cancer of the breast, lung, female genital tract, gastrointestinal tract, male genitourinary tract, leukemias and lymphomas, skin, head and neck, and cancer control. Panel discussions will be held on cancer etiology, cancer pathogenesis and spread, and cancer therapy. Other papers will be given concerning frontiers in biology and cancer research and care of the advanced cancer patient.

For further information, doctors interested may write to the American Cancer Society, Inc., Medical Affairs Department, 521 West 57th Street, New York 19.

Congress of Physical Medicine Scheduled August 21-26 in Washington

Theme of the third International Congress of Physical Medicine in Washington, D. C., on August 21-26, is the diagnosis, prevention, and treatment of physiatric diseases and disabilities.

Divisions of the theme to be considered are (1) diseases of the skeletal muscles, (2) arthritis and collagen diseases, (3) neuromuscular and musculoskeletal systems, (4) after-care of acute trauma to neuromuscular and musculoskeletal systems, (5) congenital defects causing physical disability, (6) scoliosis, (7) after-care of amputations, and (8) geriatrics.

More than 70 papers will be presented at the meeting which also will include 18 scientific sessions, an extensive program of scientific movies, a full complement of scientific and technical exhibits, and business sessions.

The Johnson Stanley Coulter Lecture will be delivered by Dr. Philippe Bauwens, London, whose subject will be "Electrodiagnosis Revisited."

International Cripple Society Meets August 28 in New York

August 28 through September 2 have been designated as meeting dates of the eighth World Congress of the International Society for the Welfare of Cripples in New York's Waldorf-Astoria Hotel.

For the first time, the meeting is being held outside of Europe. The National Society for Crippled Children and Adults will be host. More than 20 committees have been working 3 years under the leadership of Dr. Howard A. Rusk, Congress president and director of the Institute of Physical Medicine and Rehabilitation at New York University Medical Center, and Lawrence J. Linck, chairman of the Congress Committee.

The Congress is expected to draw more than 5,500 physicians, nurses, therapists, educators, scientists, employers, industrialists, government officials, and voluntary agency leaders representing more than 70 countries.

Theme of the meeting is "Rehabilitation and World Peace."

Otolaryngologic Assembly in September

The University of Illinois College of Medicine's Department of Otolaryngology is offering an intensive postgraduate basic and clinical program for practicing otolaryngologists, September 24-30, in Chicago. The assembly offers a compact program of 1 week of daytime and evening sessions.

It is designed to bring to specialists a wide variety of current advances in management, therapy, and philosophies. Review of basic morphologic features also is included by means of laboratory demonstrations, dissection, and protection, all augmented by visual aids.

Panel programs have been designed to bring out special features of otologic and reconstructive surgery and tumors of the head and neck. Luncheon chats are an important part of the daily program.

Interested physicians should write directly to the Department of Otolaryngology, University of Illinois College of Medicine, 1853 West Polk Street, Chicago 12.

Fall Clinical Conference Set August 20 in Wichita Falls

The North Texas-Southern Oklahoma Fall Clinical Conference will be held August 20 at the Wichita Falls Country Club in Wichita Falls.

Speakers include Dr. Herbert C. Miller, professor of pediatrics at the University of Kansas Medical Center whose topic is "Respiratory Distress Syndrome and the Newborn"; Dr. Franz Joseph Ingelfinger, professor of medicine at Boston University School of Medicine, "Dysphagia"; Dr. John Blalock of the Department of Surgery at the Ochsner Clinic, New Orleans, "Inferior Vena Cava Ligation in Cases of Thrombo-embolic Disease."

Jaundice will be the subject of the afternoon seminar. Also featured at the meeting will be technical exhibits and a roundtable luncheon with a question and answer period.

Registration fee for the conference is $8.

For Postgraduate Assembly: Medical Discussions in Depth

The Postgraduate Medical Assembly of South Texas has scheduled medical discussions in depth, symposiums followed by panel discussions, when it meets July 18-20 at the Shamrock Hilton Hotel in Houston.

Twenty-one out-of-state speakers will be on the program, which is divided into interests of four major sections: medicine, surgery, obstetrics and gynecology, and ophthalmology and otolaryngology.

Requests for information may be made to the Executive Committee, 412 Jesse H. Jones Library Building, Houston 25.

Pan-Pacific Surgical Association Slates Fall Hawaii Session

The eighth Congress of the Pan-Pacific Surgical Association will be held in Honolulu, September 27-October 5, with an outstanding scientific program slanted to be of interest to all physicians. Ten surgical specialty sections are held simultaneously.

Further information and brochures may be obtained by writing to Dr. F. J. Pinkerton, director general of the Association, Suite 230, Alexander Young Building, Honolulu 13.

Southern Trudeau Society to Meet

The 1960 annual meeting of the Southern Trudeau Society and the Southern Tuberculosis Conference will be held September 14-16 at the Hotel Francis Marion in Charleston, S. C.

Those interested in obtaining further information may write Mr. Judson M. Allred, Jr., P. O. Box 9865, Jackson, Miss.

At the end of 1959, the Joint Commission on Accreditation of Hospitals had accredited 3,668 hospitals. Although this was 228 fewer than in 1958, because of the withdrawal of Canadian hospitals, there was a net gain in the United States of 95 hospitals.

EDUCATION

Cunningham Fellowship To Be Initiated in Fall Term

Latest reports show that more than $4,000 has been contributed to the Harriet Cunningham Memorial Graduate Fellowship in Medical Writing at the University of Texas.

Established in memory of Miss Cunningham, the late managing editor of the *Texas State Journal of Medicine* who died May 9 in Austin, the fellowship will provide $250 each year beginning this fall for a qualified graduate student at the University.

The fellowship was created to give an opportunity to a young person of exceptional ability to gain a background for medical writing and to secure some experience in this field as a part of an educational program. A main objective is to focus attention on the challenge and opportunity in the field of medical writing for journalism students and for individuals in other liberal arts areas.

The student selected for the fellowship will be required to take 30 semester hours of courses for his master's degree. This may include a 3 hour senior-graduate course in article writing and the student may concentrate on writing articles related to medical fields. He also may enroll in a research or work-project conference course, which would be planned in cooperation with Texas Medical Association. For thesis work (6 hours credit) the fellowship recipient would be expected to select a research area connected with medical writing. Eighteen remaining semester hours would be devoted to broadening and strengthening the student's background and might include sociology, psychology, political science, and science.

Individuals interested in applying for the scholarship may address Dr. DeWitt C. Reddick, School of Journalism, The University of Texas, Austin 12, or Mr. C. Lincoln Williston, Texas Medical Association, 1801 North Lamar Boulevard, Austin 14.

Postgraduate Courses

The Internal Medical Audit, Denver, August 10-12.—The Office of Postgraduate Medical Education at the University of Colorado Medical Center, Denver, is presenting this course in response to the increasing interest in the medical audit. Subjects to be discussed will include many practical and experience-tested aspects of the field. One half-day of the course will be devoted to separate concurrent sessions dealing with the special problems and methods of auditing the four major specialities—medicine, surgery, pediatrics, and obstetrics. The course is being offered primarily for hospital chiefs of service and other staff physicians, but a limited number of hospital administrators may attend. Tuition fee for the course is $35 and includes a nonrefundable registration of $5. The course is acceptable for American Academy of General Practice Category I credit for the number of hours attended. Further information may be obtained from the Office of Postgraduate Medical Education, the University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20.

Pediatrics, Estes Park, Colo., September 1-6.—In two parts, this 5 day course on pediatrics is being presented by the University of Colorado School of Medicine, the Department of Pediatrics, and the Office of Postgraduate Medical Education of the University of Colorado Medical Center. The first part of the course, September 1-3, will

be devoted to Clinical and Research Advances in Pediatrics, and the second part on September 5 and 6, will be on Child Guidance. The entire course is to be held at the Stanley Hotel in Estes Park, Colo., and regularly scheduled bus service will be available from Denver to Estes Park. Tuition fee is $60 for the entire course and $30 for either of the two parts. Also required is a $10 nonrefundable registration fee. Further information may be obtained from the Office of Postgraduate Medical Education, University of Colorado Medical Center, 4200 East Ninth Avenue, Denver 20.

Hematology and Radioisotopes, Philadelphia, September 19-23.—The American College of Physicians announces its Course I in Hematology and Radioisotopes to be held at the Ohio State University College of Medicine in Columbus, Ohio, September 19-23. Tuition fee for members is $60 and nonmembers, $80. Registration forms and requests for information may be obtained by writing Dr. Edward C. Rosenow, Jr., Executive Director, American College of Physicians, 4200 Pine, Philadelphia.

Cancer and the Internist, New York, October 10-14.—Course No. 2, Cancer and the Internist—1960 Concepts will be sponsored by the American College of Physicians, October 10-14, at the Memorial Center, Sloan-Kettering Institute for Cancer Research in New York. Tuition fee for members is $60 and nonmembers, $80. Those interested may obtain registration forms and further information from Dr. Edward C. Rosenow, Jr., Executive Director, American College of Physicians, 4200 Pine, Philadelphia.

Baylor University College of Medicine

A unique joint appointment designed to promote a closer working relationship in the medical sciences between Baylor University College of Medicine and the College de France in Paris was announced by Dr. Stanley W. Olson, Dean of the College of Medicine in the Texas Medical Center.

The joint appointment provides that as of July 1, Dr. Roger Guillemin, associate professor of physiology at Baylor, also will become associate director of the Institute for Experimental Endocrinology at the College de France. Under this arrangement, made possible by a grant of $5,000 from the Houston Endowment, Inc., Dr. Guillemin will divide his time between Houston and Paris.

Establishment of the Peggy Ann Efron Student Research Fellowship in Hematology in the Department of Pediatrics at Baylor was announced recently by Dr. Russell J. Blattner, professor and chairman of the Department of Pediatrics. The fellowship is made possible by a gift of $600 from the Sisterhood Temple Beth Israel and is a memorial to the daughter of Meyer Efron, Peggy Ann Efron, who died of leukemia several years ago.

A gift of $300,000 by *Mr. and Mrs. J. Newton Rayzor* and their family for a new Student Center at Baylor University College of Medicine in the Texas Medical Center, Houston, has been announced by Dean Stanley W. Olson. The building will include a two-level, glass-enclosed dining and recreation area with landscaped courtyards, a faculty dining room, exhibit area, student and faculty meeting places.

Dr. George L. Jordan, Jr., associate professor of surgery at the college, has been selected as recipient of a $20,000 Lederle Medical Faculty Award for 1960-1961. He is the first member of the Baylor faculty to receive one of the Lederle awards. Dr. Jordan has published more than 50 original papers dealing with research on atherosclerosis, gastric resection, and the effect of anticancer substances.

University of Texas Medical Branch

Dr. Robert H. Berger, Lubbock, May graduate of the University of Texas Medical Branch, Galveston, was named first recipient of the Gold Headed Cane Award during commencement exercises. Chosen by his classmates as the "graduate who best exemplified the qualities of the physician who will put the welfare of his patient first at all times," Dr. Berger was given the cane by Dr. William Kerr, retired chairman of the University of California School of Medicine.

The University of Texas Medical Branch is the third school in the nation to revive the tradition instigated in England in 1714. The teak wood cane, bearing the words of "Humility, Humanity, Fidelity" and topped by a solid gold head of riding-crop style, is to be kept by Dr. Berger 1 year before he turns it over to his successor in 1961. Each year a gold band with the name of the new winner inscribed on it will be added to the shaft.

The ceremony and award were adopted at the suggestion of Dr. Charles T. Stone, Sr., professor of internal medicine.

Two other students, Dr. Charles Stevens of LaMarque, and Dr. Thomas E. Taylor of Quanah, were honorable mentions for the award.

Other United States schools that offer the cane are the University of California and the University of Colorado.

Dr. Frank Harrison, professor of anatomy and assistant dean of the University of Texas Southwestern Medical School, Dallas, was speaker May 16 at the University of Texas Medical Branch, Galveston. His topic was "Present Concepts of Neuron Function."

Dr. Truman G. Blocker, Jr., will become chairman of the Department of Surgery of the University of Texas Medical Branch, effective July 1, succeeding Dr. Robert M. Moore. Dr. Moore will continue to serve as an active full professor in the department.

Dr. Blocker has been chief of the Medical Branch division of plastic and maxillofacial surgery since 1945.

Guest speakers at the Medical Branch in recent weeks have included Dr. Andrew Ranier, pathologist, Lake Charles, La.; Harold G. Cassidy, Ph.D., professor of chemistry, Yale University; Dr. David E. Rogers, professor of medicine, Vanderbilt University; and Dr. R. F. Ogilvie, reader in pathology, University of Edinburgh Medical School, Scotland.

Dr. Leon Sokoloff, research pathologist at the National Institute of Arthritis and Metabolic Diseases, Bethesda, Md., spoke on "The Biology of Degenerative Joint Disease," which was a Sigma Xi lecture, on March 29 at the University of Texas Medical Branch, Galveston.

Obstetrics-Gynecology Board Certifies 12

Twelve Texas doctors are among those recently examined and certified by the American Board of Obstetrics and Gynecology.

They include Dr. Robert Henry Benbow, Bryan; Dr. Donald P. Bernard, United States Naval Hospital, Corpus Christi; Dr. Andy James Bowden, Jr., Fort Worth; Dr. Francis Waring Burke, United States Naval Hospital, Corpus Christi; Dr. Robert Vernon Colpitts, Houston; and Dr. Joe Dudley Crawford, Jacksonville.

Also Dr. Harold Irvin Daily, Houston; Dr. Charles Frederick Ford, United States Air Force Hospital, Reese Air Force Base, Lubbock; Dr. James Henry Goodson, Dallas; Dr. Michael Kurilecz, Dallas; Dr. Donald Edward McGuire, Dallas; and Dr. John B. Norton, United States Air Force Hospital, Sheppard Air Force Base, Wichita Falls.

Groundbreaking Ceremonies Held For New Baylor Building

Groundbreaking ceremonies for the M. D. Anderson Basic Science Building and the Jesse H. Jones Clinical Research Building in the Texas Medical Center, Houston, were held on May 21, and ceremonies for the Jewish Medical Research Institute were held on June 7.

Provided by the Texas Medical Center for the use of Baylor University College of Medicine, basic science and clinical research buildings are being built from grants of $1,000,000 each by the Houston Endowment and the M. D. Anderson Foundation, and matching funds from the Health Research Facilities of the National Institutes of Health.

Both the basic science building and the clinical research building will contain five stories plus basement and subbasements. Each will have about 66,000 square feet of floor space. Plans provide for 150 laboratories and offices in each building. Architecture will be modern with exposed structural beams and curtain wall construction.

The basic science building will adjoin the research building, and will provide facilities for the departments of dermatology, anatomy, genetics, biostatistics, pharmacology, medicine, and surgery. It will provide for research activities in the latter two departments. The clinical research building will house the departments of obstetrics and gynecology, neurology, pathology and neuropathology, pediatrics, internal medicine, and visual education. It will be used also for research in studying the clinical problems of medicine in the laboratory.

The groundbreaking ceremonies marked the beginning of the expansion program which also will include eventually the Rayzor Student-Faculty Center.

Unique Foreign Fellowships To Send Students to Remote Areas

The Association of American Colleges has announced the establishment of a unique fellowship program designed to further medical education by sending future doctors to remote areas of the world.

The 3 year program, established under a $180,000 grant from Smith Kline and French Laboratories, is open to all medical college students who have completed their third year of study. Scheduled to begin this summer, the program will permit an average of 30 students to participate each year. They will spend an average of 12 weeks working in the more primitive regions of the world, learning the mores and ethical backgrounds of other peoples and representing the United States as unofficial ambassadors.

Awards will be made on the basis of the applicant's ability and objectives. In some cases, a contribution will be made for the student's spouse if this seems desirable in terms of the objectives of the trip.

Further information may be obtained from the Association of American Medical Colleges, 2530 Ridge Avenue, Evanston, Ill.

Houston VA Hospital Studies Drugs

The Houston Veterans Administration Hospital is among 37 such institutions across the nation participating in a controlled study of use of three newer antidepressive drugs in treatment of mental and emotional illness. The drugs are imipramine, isocarboxazid, and dextro-amphetamine sulphate.

Arthritis Fellowships, Awards Available for Three Groups

The Arthritis and Rheumatism Foundation is offering predoctoral, postdoctoral, and senior investigatorship awards in the fundamental sciences related to arthritis for work beginning July 1, 1961. Deadline for applications is October 31, 1960.

These awards are intended as fellowships to advance the training of young men and women of promise for an investigative or teaching career. They are not in the nature of grants-in-aid in support of a research project.

Predoctoral fellowships are limited to students who hold bachelors' degrees, stipends to range from $2,000 to $3,000; postdoctoral fellowships are limited to applicants with degrees of doctor of medicine, doctor of philosophy, or the equivalent, stipends to range from $5,000 to $7,000 per year; and senior investigator awards are made to candidates holding or eligible for a faculty rank such as instructor or assistant professor, stipends to range from $7,000 to $10,000 per year.

For further information, inquiries may go to the Medical Director, Arthritis and Rheumatism Foundation, 10 Columbus Circle, New York 19.

Obstetrics and Gynecology Board Schedules Part I Examination

The next scheduled American Board of Obstetrics and Gynecology Examination (Part I), written, will be held in various cities of the United States, Canada, and military centers outside the Continental United States on January 13, 1961.

Candidates submitting applications in 1960 for the 1961 examinations are not required to submit case reports as previously required to complete the part 1 examinations of this board. In lieu of this requirement, new candidates are required to keep in their files a duplicate list of hospital admissions as submitted with their application, for submittal at the annual meeting in Chicago should they become eligible to take the Part II (oral) Examinations.

Reopened candidates will be required to submit case reports for review 30 days after notification of eligibility. Those scheduled for Part I and candidates resubmitting case reports are required to submit case reports prior to August 1 each year.

Current bulletins may be obtained by writing to: Dr. Robert L. Faulkner, 2105 Adelbert Road, Cleveland 6.

Requests for reexamination in Part II now are being accepted. All candidates are urged to make such application at the earliest possible date. Deadline for receipt of applications is August 1.

The following change in requirements for certification was made by the members of the American Board of Obstetrics and Gynecology at the recent annual meeting in Chicago.

"A Resolution was passed at the recent annual meeting of this Board which eliminates the submission of Case Reports as part of the Part I Examination. It is required, however, that each candidate eligible to take the Part II Examination bring to the place of examination, a duplicate list of Hospital Admissions as submitted with his or her application. *This change in requirements is not retroactive and therefore applies to candidates making application for the 1961 examinations.*"

Members of the board resolved that applications for appraisal of incomplete training will no longer be accepted for review by the Residency Review Committee.

Personals

Dr. John J. Andujar, Fort Worth, was in Moscow July 5 as an official delegate of the American Society of Clinical Pathologists to deliver a scientific report at the Herzen Institute. Dr. Andujar studied the Russian language 10 months prior to his trip so that he would be able to present his paper in the Soviets' own tongue. In addition to his Russian talk, Dr. Andujar spoke in Spanish at the meeting of the International Society of Pathologists in Madrid, prior to his Russian appearance.

Dr. N. L. Barker, Paris, was featured in a recent biography, "Paris Profiles," in the *Paris News.*

Dr. Harris Busch, Chicago, has been appointed professor of pharmacology and chairman of the Department of Pharmacology at Baylor University College of Medicine, Houston. Dr. Busch has been professor of pharmacology at the University of of Illinois College of Medicine, Chicago.

Dr. John McGivney, instructor in surgery at the University of Texas Medical Branch, Galveston, presented an illustrated lecture on "A Simple Procedure for the Correction of Postoperative Anal Stenosis" at the twelfth International Surgical Conference held in Rome in May.

Dr. and Mrs. C. P. Schenck, Fort Worth, were honored May 18 as that city's outstanding senior citizens for 1960 by the Women's Civic Club Council. First to receive the award jointly, the couple was feted at a dinner at the Elks Club.

Dr. George R. Herrmann, professor of medicine at the University of Texas Medical Branch in Galveston and director of Cardiovascular Research Laboratory and Heart Station, recently was made an honorary fellow of the American College of Cardiology.

Dr. Ben M. Primer, Austin, was spotlighted in an article in the *Austin Municipal Review,* publication of the City of Austin, concerning his job as director of the Austin-Travis County Health Unit and city health officer.

Dr. James A. Knight, Houston, was named assistant dean of Baylor University College of Medicine, effective July 1.

Dr. Ray K. Daily of Houston recently attended a meeting of the International College of Surgeons in Rome, Italy.

Drs. Michael E. DeBakey and *George C. Morris, Jr.,* of Houston, were awarded first prize for the scientific motion picture, "Fusiform Aneurysm of Aortic Arch, Resection and Replacement with Dacron Graft," presented at the twenty-sixth annual meeting of the American College of Chest Physicians on June 11.

Dr. and Mrs. Michael E. DeBakey, Houston, recently were in Moscow, where Dr. DeBakey attended the Seventh Annual Surgical Congress meeting there.

Five Texas Laymen Honored by Physicians

The Southern Medical Association honored five Texas laymen with associate memberships during the 1960 annual session of the Texas Medical Association.

Voted associate membership awards by the Southern Medical Association Council, its governing body, were C. Lincoln Williston, executive secretary, and Donald M. Anderson, assistant executive secretary, of the Texas Medical Association, Austin; Millard J. Heath, executive secretary of the Dallas County Medical Society; Philip R. Overton, general counsel of the Texas Medical Association, Austin; and S. E. Cockrell, Jr., executive secretary of the Bexar County Medical Society, San Antonio.

The honors were given "for meritorious service to the medical profession or to humanity by a layman."

Rouse Speech Cites Forand Bill Faults

Dr. Milford O. Rouse, Dallas, vice speaker of the American Medical Association's House of Delegates and a past president of the Texas Medical Association, reiterated the dangers of federal intervention in medical care for the aged when he addressed persons attending a recent 2-day conference sponsored by the American Medical Association's Committee on Aging in New Orleans. Co-sponsors were the state medical societies of Texas, Arkansas, Louisiana, and Mississippi.

He voiced three specific objections to the enactment of Forand-type legislation: (1) already over-crowded hospitals would be swamped if their doors were thrown open to all old people regardless of whether or not they needed hospital care, (2) the cost to taxpayers would be extensive, initial cost alone of putting the Forand bill into operation exceeding $2,000,000,000, and (3) "the grave fear that once government guarantees services it cannot itself provide, it tends to control those who do provide the services. This is a long way of saying he who pays the piper calls the tune."

America's aging population desires to be independent and self-sufficient, Dr. Rouse pointed out. "Senior citizens want only a hand, not a handout."

Alcoholic Rehabilitation Program Begun by State at Huntsville

Rehabilitation counselor Nick Carter, Lake Jackson, has assumed his duties with the Texas Department of Corrections, to initiate a state program for alcoholic rehabilitation.

Created by the fifty-sixth Legislature to facilitate and coordinate activities of outside Alcoholics Anonymous groups now working in 12 units of the prison system, the position was filled for the first time in January when Mr. Carter took office.

Stationed in Huntsville, the counselor is responsible to the director of the Department of Corrections. Appropriations supporting the project was made to the Department of Corrections to be spent with the advice of the Texas Commission on Alcoholism. Policy and procedure were worked out together by representatives of the two agencies.

Artificial Insemination Increasing New Digest Asserts

More and more women are having babies by artificial insemination, and increasing numbers of physicians are engaging in this practice, according to an article in *Medicolegal Digest,* a new monthly launched May 20 for the medical profession.

Because of the secrecy surrounding it, very little is known about the extent of this medical procedure, first performed in this country in 1886. The article estimates that "in the United States today there are perhaps 50,000 or more individuals conceived from the semen of unidentified donors, and 1,000 to 2,000 such conceptions each year." Approximately 5,000 physicians in America engage in artificial insemination, and many of the donors are married interns and residents, who are paid between $10 and $25 per semen specimen.

Donor insemination is cloaked in secrecy, points out the author, Milton Golin, because there is no basis anywhere in the world for definitely establishing such points of legality as child custody, legitimacy of the birth, and whether the practice constitutes adultery.

MEMORIAL LIBRARY

Books Newly Acquired

Advances in Pediatrics: vol. 11, Chicago, Year Book Publishers, 1960.

Bedichek, Roy: *Sense of Smell,* Garden City, Doubleday, 1960.

Burch, George E., and Winsor, Travis: *Primer of Electrocardiography,* ed. 4, Philadelphia, Lea and Febiger, 1960.

Castellani, Aldo: *A Doctor in Many Lands, The Autobiography of Aldo Castellani,* Garden City, Doubleday, 1960.

Clark, Wallace and Anne: *Guide to Medical Terminology,* Philadelphia, F. A. Davis Co., 1956.

Feldman, Sandor S.: *Mannerisms of Speech and Gestures in Everyday Life,* New York, International, 1959.

Leavell, Byrd S., and Thorup, Oscar A., Jr.: *Fundamentals of Clinical Hematology,* Philadelphia, W. B. Saunders, 1960.

Moyer, John H., and Fuchs, Morton: *Edema, Mechanisms and Management,* Philadelphia, W. B. Saunders, 1960.

Pack, George T., and Ariel, I. M.: *Treatment of Cancer and Allied Diseases: Tumors of the Nervous System,* vol. 2, New York, Hoeber, 1959.

Pack, George T., and Ariel, I. M.: *Treatment of Cancer and Allied Diseases: Tumors of the Head and Neck,* vol. 3, New York, Hoeber, 1959.

Progress in Allergy: vol. 5, Basel, Switzerland, Karker, 1958.

Quimby, Edith H.; Feitelberg, S.; and Silver, S.: *Radioactive Isotopes in Clinical Medicine,* Philadelphia, Lea and Febiger, 1958.

Rutledge, Hames Hale: *Concise Encyclopedia of Modern Surgery,* Philadelphia, Chilton, 1960.

Russian Scientific Translation Program: *Central Nervous System and Human Behavior,* Washington, United States Department of Health, 1960.

Spiegelman, Mortimer: *Ensuring Medical Care for the Aged,* Homewood, Ill., Richard D. Irwin, Inc., 1960.

Staff of the Mount Sinai Hospital, edited by Alan F. Guttmacher: *Medical, Surgical and Gynecological Complications of Pregnancy,* Baltimore, Williams and Wilkins, 1960.

Transactions of the Ophthalmological Society of the United Kingdom: vol. 79, 1959, Churchill, 1960.

Viets, Henry R., and Schwab, Robert S.: *Thymectomy for Myasthenia Gravis,* Springfield, Ill., Charles C Thomas, 1960.

Year Book of Dermatology, 1959-60, Chicago, Year Book Publishers, 1960.

Year Book of Neurology, Psychiatry and Neurosurgery, Chicago, Year Book Publishers, 1960.

Library Receives Russian Journals

The Memorial Library receives the following Russian journals which are printed entirely in English. Illustrations are clear and well reproduced. Charts and graphs are given adequate spacing for legibility and easy reading.

Abstracts of Soviet Medicine, Part A—Basic Medical Sciences; Part B—Clinical Medicine.

Biophysics.

Sechenov Physiological Journal of the U.S.S.R.

Pavlov Journal of Higher Nervous Activity.

Journal of Microbiology, Epidemiology and Immunobiology.

Problems of Oncology.

Problems of Virology.

Problems of Hematology and Blood Transfusion.

Biochemistry.

Bulletin of Experimental Biology and Medicine.

These journals come through the courtesy of the National Institutes of Health.

Gifts to the Library

Dr. Mathis W. Blackstock, Austin, 96 journals.

Dr. George E. Clark, Jr., Austin, 101 journals.

Mrs. W. Daly, Knoxville, Tenn., 70 books.

Dr. Morris Davidson, Austin, 1 bulletin.

Dr. Robert F. Ellzey, Austin, 9 journals.

Drs. Sigman W. Hayes and Mathis W. Blackstock, Austin, 93 journals.

Dr. Maurice Jacobs, Austin, 7 journals.

Dr. J. Edward Johnson, Austin, 39 reprints, 2 journals, 1 print, and 19 bulletins.

Dr. Sam N. Key, Jr., Austin, 1 book, 14 journals.

Dr. Virgil B. Lawlis, Austin, 24 journals.

Drs. Georgia and Carey Legett, Austin, 14 journals.

Philip R. Overton and Pat Bailey, Austin, 17 journals, 1 bulletin.

Dr. Howard O. Smith, Marlin, 50 journals, 62 books, 12 bulletins, and 8 reprints.

Dr. Teddy M. Sousares, Austin, 25 journals, 3 books.

Dr. Samuel P. Todaro, Austin, 1 journal.

Dr. B. O. White, Austin, 18 journals.

Archives of Neurology Needed

The Memorial Library of the Texas Medical Association needs, for binding purposes, volume 1 of the 1959 AMA Archives of Neurology. Miss Pauline Duffield, Librarian, would appreciate any owner of this volume donating it to the Library. Donations are to be sent to Miss Duffield, Texas Medical Association, 1801 North Lamar Boulevard, Austin.

Book Notes

Leukemia

WILLIAM DAMESHEK, M.D., Professor of Medicine, Tufts University School of Medicine; Senior Physician and Director, Blood Research Laboratory, New England Center Hospital, Boston, and FREDERICK GUNZ, M.D., Ph.D., Hematologist, Christchurch Hospital, Christchurch, New Zealand. 420 pages. $15.75. New York and London, Grune and Stratton, 1958.

Being an amateur photographer, my first notice would be naturally the excellence of reproductions of radiographs, the line drawings, microphotographs, and actual photographs of the patients with different manifestations of the disease itself.

The discussion of what a physician should tell the patient is a portrayal of experience that all of us have gone through, thought about, and dwelt upon many times. What it really boils down to is: First, completely know

your patient and then tell him as much as it is safe to do, but above all do not destroy all his vestiges of hope for the future; at the same time completely inform a responsible relative.

The discussions of diagnosis, prognosis, treatment, and special features of the disease are a rich addition to the literature. The representative and special case studies presenting the many facets, problems, and methods of management of this disease make it a valuable addition. Finally, as a nice touch enhancing the value of this book, are its color plates depicting polycythemia vera, myelosclerosis with myeloid metaplasia, chronic granulocytic leukemia, and others.

The comprehensive treatment as to classification, etiology, pathology, course, special pathology, symptomatology, clinical course, special features, and laboratory diagnosis as well as treatment makes this publication a reference of practical help that anyone who deals with this condition will value on his working library shelf. The extensive bibliography is a valuable contribution in this work.

—Joe C. Rude, M.D., Austin.

Lipidoses—Diseases of the Intracellular Lipid Metabolism

SIEGFRIED J. THANNHAUSER, M.D., Ph.D., Hon. M.D., Universities of Freiburg, Munich, Dusseldorf, Clinical Professor of Medicine Emeritus, Tufts College Medical School, Boston; Consulting Physician, Pratt Diagnostic Hospital, New England Medical Center, Boston. ed. 3. 590 pages. $19.75. New York and London, Grune and Stratton, 1958.

This 590 page exhaustive review of the disorders of fat metabolism is complete and thorough. The pathology of all forms of fat metabolism is discussed very adequately. Completely reviewed are all the varied and sundry disorders which are characterized by an abnormal increase in serum and intracellular and extracellular depositions of cholesterol, neutral fat, cerebrosides, and other similar substances.

The secondary xanthomatoses are adequately discussed. This includes hypercholesteremic xanthomatoses secondary to liver disease, thorazine hypersensitivity, pigment cirrhosis, hemochromatosis, hypothyroidism, and lipoid nephroses.

Separate discussions are given of the Schuller-Christian syndrome (cholesterol at fault), Gaucher's disease (cerebrosides at fault), and Niemann-Pick's disease (sphingomyelinosis—the metabolic culprit). Tay-Sachs disease or infantile amaurotic idiocy is also discussed as is the Hurler syndrome, which all authors do not accept as a true lipidosis.

This book is written in great detail and contains a tremendous bibliography of more than 1,292 listings. Therefore, it is an excellent reference book and a book to peruse carefully, rather than one just for casual reading. There are occasional misspellings which should not detract from the value of this classic.

—Rider E. Stockdale, Jasper.

Medical Department, United States Army: Surgery in World War II—Neurosurgery, Vol. I

Prepared and published under the direction of MAJOR GEN. S. B. HAYS; Editor in Chief, COL. JOHN B. COATES, JR., MC; Office of the Surgeon General, Department of the Army, Washington, D. C., 1958.

This volume had particular significance to the reviewer because of his previous personal contact with Dr. Barnes Woodhall while serving as instructor in radiology at Duke Hospital and Medical School just before the war, and with Dr. R. Glen Spurling during World War II. It was his privilege for a short time to serve as radiologist in the 158th General Hospital and to witness the busy neurosurgical service while the regular radiologist was on sick leave.

This monumental collection of material contains information not only for the handling of immediate injuries but for the handling of the many sided problems in restoring these patients to useful lives after injury. Absorbing the findings recorded here could obviate many mistakes in the future handling of these casualties on a large scale.

Illustrations are well reproduced and are instructive in connection with material presented. Particularly impressive was the material dealing with injuries to the ventricular system and the manner in which it was handled. The sections on post-traumatic epilepsy, the pathology of missile injuries, and speech disorders and defects are worthy of mention.

Last, but not least, is the special attention given to complete history and physical forms in Appendix C. If one read nothing else in this book, it would be worth while to spend some time on it, for it will make him a better physician.

In the reviewer's opinion, the decision to collect all neurosurgical cases in special centers was one of the really great contributions in World War II. It made possible the collection of well-trained teams and it is an important factor in the specialty of neurosurgery, coming into full flower as a medical specialty with recognition and an established place in medicine.

—Joe C. Rude, M.D., Austin.

Callander's Surgical Anatomy

BARRY J. ANSON, Ph.D. (Med. Sc.), Chairman, Department of Anatomy; ROBERT LAUGHLIN REA, Professor, Northwestern University Medical School; WALTER G. MADDOCK, M.D., F.A.C.S.; and EDWARD S. ELCOCK, Professor of Surgery, Northwestern University Medical School. ed. 4. 1157 pages. $21. Philadelphia and London, W. B. Saunders Company, 1958.

The authors have not changed the pattern of presentation in this edition from that which was successful in former ones.

The anatomy is regional and, in general, utilitarian or directed to the understanding of surgical procedures and their consequences. In the surgical parts the rationale rather than details of procedure is stressed. Several specialists have assisted with the text in their various fields.

Material has been brought up to date, numerous references are given, and the sources of borrowed illustrations are given. Particularly valuable is the emphasis upon the troublesome variations encountered by the surgeon. These descriptions, together with their frequency, are largely taken from the works of Dr. Anson and his colleagues. Frequent references are made also to anastomotic connections of blood vessels.

New material has been added throughout, such as that for the abdominal wall, vessels of the liver, and lymphatics of the intestine. Enlargement of various sections has been made, as with the parotid and mammary glands. To the preceding edition, 82 pages and 118 figures have been added. In general, the illustrations are very good although those for the female are less satisfactory. Changes in standard anatomical nomenclature adopted by the International Congress of Anatomists have not been used.

—Raymond F. Blount, M.D., Galveston.

501 Questions and Answers in Anatomy

STANLEY D. MIROYIANNIS, B.S., M.A., Ph.D., F.A.A.A.S., F.I.A.S., Professor of Anatomy and Chairman of the Department, Still College. 332 pages. $5. New York, Washington, Hollywood, Vantage Press, Inc., 1959.

AMA House of Delegates Favors Texas Resolutions

Views of Texas physicians, as reflected by resolutions introduced by delegates from the Texas Medical Association, were favorably received by the American Medical Association House of Delegates at the annual meeting at Miami Beach on June 13-17.

The resolutions called for actions on the following variety of subjects of current medical interest: (1) food and color additives; (2) opposition to a proposed federal program of international medical research; (3) basic reform of federal tax rates and measures; (4) efficiency study of hospital records; (5) establishment of an AMA program of group disability insurance; and (6) appeals by physicians to the AMA Judicial Council.

As evidence of the favorable support accorded these resolutions, all but one were either passed as presented, approved in principle, or endorsed by substitute resolution. The first four resolutions were introduced by Texas delegates before the AMA as a result of action taken by the Texas Medical Association House of Delegates in April at Fort Worth.

Specific action by the AMA House of Delegates on Texas resolutions follow:

1. *Color and food additives.* The House approved a resolution which calls for the AMA to review existing and federal legislation dealing with food and color additives, and then support measures which are scientifically sound and practical, and in the public interest. The Texas resolution pointed out that there has been considerable public and scientific concern, as well as controversy, in regard to the addition of cancer-causing chemicals and other hazardous substances to foods. The resolution also expressed the view that it is in the public interest to establish criteria for eliminating hazardous substances from food as well as criteria which permit adequate scientific appraisal of these effects upon humans.

The AMA previously had not expressed itself on a food additive bill which was enacted into law by the Eighty-fifth Congress, or two bills which regulate the use of color additives introduced by Senator Lister Hill (D., Ala.) and Representative James J. Delaney (R., N.Y.) in the current Eighty-sixth Congress. The Texas resolution stated that the AMA is looked upon for leadership, for guidance, and for counsel in scientific matters related to medicine, and that the AMA should investigate and express itself on these measures.

2. *Opposition to a proposed federal program of international medical research.* The Texas resolution called for opposition to Senate Joint Resolution 41 and House Joint Resolution 649 which it termed as "unnecessary, impractical, and undesirable as a new avenue of foreign aid." At the same time, the Texas resolution asked the AMA to heartily reiterate its support of medical research by an individual or organization which has available resources and personnel to carry out planned, worthwhile investigations.

The Texas resolution pointed out that S. J. R. 41 and H. J. R. 649 "could very well represent an unwise and wasteful expenditure of the taxpayers' money," and would be an additional appropriation for foreign aid. Texas delegates testified that these proposals come at a time when Congress is concerned with the scope and magnitude of foreign aid, and sound leaders of the nation's economy have warned that it is imperative to control federal expenditures and balance the budget.

The AMA House voted to take no action on the resolution at this time, however, pointing out that this resolution has been and still is under study by the Council on Legislative Activities.

3. *Basic reform of federal tax rates and measures.* The Texas resolution asked the AMA to support the Herlong-Baker bills in Congress (H. R. 3000 and H. R. 3001) which would provide for basic reform to federal tax rates and methods, and remove "tax blocks to progress." The provisions of these bills would (a) moderate personal and corporate income taxes over a 5 year period to a top rate of 47 per cent; (b) establish more realistic depreciation rules; (c) defer taxes for individuals on longterm capital gains until such time as the taxpayer "dis-invests"; and (d) reduce the rates of tax on estates and gifts.

The Texas resolution further stated that the Herlong-Baker bills would contribute to this nation's economic well-being and national security. It pointed out that the present federal tax structure stunts economic growth since it prohibits adequate capital accumulation and destroys capital once accumulated.

The House endorsed the objectives of the resolution, but expressed the view that it was more desirable for the AMA to emphasize principles rather than specific legislation. It said it was preferable to examine tax principles with reference to the health needs of the people and their rights for self determination.

The substitute resolution adopted by the House of Delegates called upon the AMA to "join in supporting the activities of other organizations seeking reform of the federal tax structure so as to return to the states and the political subdivisions their traditional revenue sources and to allow American citizens to enjoy the fruits of their labor."

The substitute resolution also made the following points: (a) the federal tax base is too broad and preempts the ability of the states to obtain revenue from many sources historically available to them; (b) as a result of this preemption, the states and their political subdivisions are finding it impossible to exercise self determination and to meet many responsibilities, including the provision of medical care for their needy and near needy citizens; (c) federal taxes rapidly are approaching or have reached the confiscatory level; and (d) as a result of the high federal tax rate and its accompanying inflation, many American citizens no longer are able to protect themselves against economic and other crises.

4. *Efficiency study of hospital records.* The Texas resolution directed the AMA to ask its appointees to the Joint Commission on Accreditation of Hospitals to urge the commission to conduct an efficiency study of present hospital records with the aim of eliminating duplication, and to call upon record management authorities outside of the health care field who are in position to contribute to the objective.

The Texas resolution pointed out that it is most desirable to continue to refine techniques and procedures with the objectives of eliminating duplication in hospital records. It acknowledged that efficiency engineers and record management experts could make a significant contribution, if called upon, to make an objective study of hospital records.

Two other similar resolutions were introduced before the AMA House, one from Missouri and the other from

the Section on Orthopedic Surgery, all for the purpose of assisting the Joint Commission on the Accreditation of Hospitals in performing its overall responsibilities. The three resolutions were considered together, and it was agreed that the study proposal would be of value and that it should be undertaken by the parent organizations of the Joint Commission.

The House subsequently adopted a substitute resolution requesting the AMA Board of Trustees to implement a study of the present policy regarding the required content and method of preparing hospital records.

5. *Establishment of an AMA program of group disability insurance.* The House of Delegates went on record favoring a Texas resolution which instructed the Board of Trustees to implement a program of comprehensive disability insurance for AMA members which would not compete with plans being offered by state and county societies. The AMA Board was directed to prepare and present a plan for consideration at the 1960 Clinical Session at Washington, D. C., in December.

Texas delegates testified before the reference committee that many physicians would welcome a group plan providing lifetime benefits from total disability resulting from sickness or accident, and embracing low premiums. They pointed out that many disability insurance programs now in force impose age limitations or drastically reduced benefits upon the older physicians and the poorer risks, thus depriving them of protection at a time when it may be needed greatly.

The Texas resolution stated that a plan of group disability insurance would represent another excellent service of the American Medical Association to its members, and that it might well eliminate many future cases of financial hardship within the ranks of the profession.

6. *Appeals by physicians to the AMA Judicial Council.* The AMA House of Delegates also was in accord with a Texas resolution which sought to reduce the period of time in which appeals to the Judicial Council may be made from 6 months to a possible 3 months following a decision by a constituent association. The House subsequently amended the AMA bylaws to stipulate that "notice of appeal shall be filed with the Judicial Council within 30 days of the date of decision by the constituent association, and the appeal shall be perfected within 60 days." However, the Judicial Council may grant an additional 30 days for perfecting the appeal for what it considers good and sufficient cause.

In testifying before the Reference Committee, the Texas delegates pointed out that 6 months was an excessive length of time, and merely acted to further postpone a final decision on matters of controversial nature. They testified that neither party would be prejudiced by a shortening of the period of time to perfect an appeal, and that the best interests of justice, the public, the appealing party, and medicine would be served by a speedy determination of the Judicial Council.

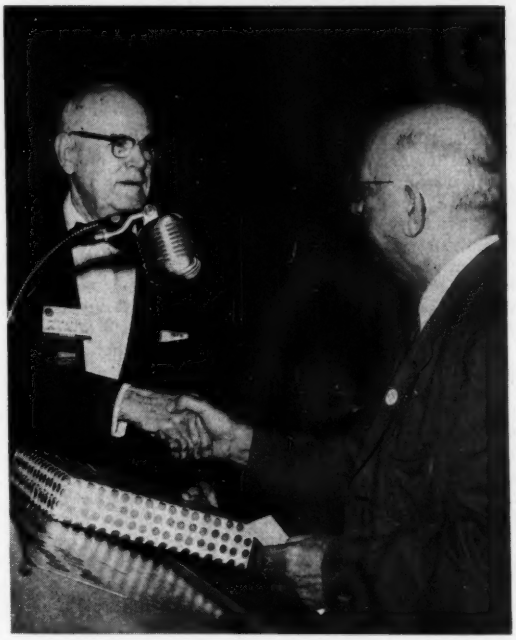
The Texas delegation actively supported a Georgia resolution which called for AMA opposition to compulsory inclusion of physicians under the retirement features of the Social Security Act. The AMA House of Delegates adopted the resolution, thus reaffirming a position which it had taken many times previously.

The House also adopted a Texas amendment, recommending that immediate action be taken by all AMA members who are in accord with this position. The amendment asked physicians to communicate immediately with their representatives and senators, as well as the President and Vice President, urging that physicians be excluded from coverage in legislation pending before the Eighty-sixth Congress.

Texan Honored at AMA Meeting

Dr. B. E. Pickett, Sr., Carrizo Springs, was honored by the House of Delegates of the American Medical Association at the annual meeting on June 13-17 in Miami Beach.

Dr. Pickett, 84, was honored upon his retirement as chairman of the AMA Council on Constitution and By-laws. He was presented with a gift on behalf of the council by Dr. Walter E. Vest, Huntington, W. Va., and was given a standing ovation by the House of Delegates.



Dr. B. E. Pickett of Carrizo Springs was congratulated before the AMA House of Delegates upon his retirement as chairman of the AMA Council on Constitution and By-Laws by Dr. Walter E. Vest, council member from Huntington, W. Va.

At 84, Dr. Pickett was introduced by Dr. Norman Welsh, Boston, Speaker of the House as "the oldest active member of the House of Delegates."

Dr. Pickett was a member of the special committee which was appointed to revise the Constitution and By-laws of the AMA in 1946. He served on that Committee for 3 years, and then completed two full terms of 5 years each on the Council on Constitution and By-laws.

Dr. Fox Appointed Member Of AMA's New Commission

Dr. Everett C. Fox, Dallas, has been appointed a member of the American Medical Association's Commission on the Cost of Medical Care.

The Commission will study pertinent aspects of medical care costs and financing mechanisms. In conducting its study, the commission will consult with providers of medical care, representatives of hospitals, and other medical facili-

ties, health insurers on prepayment plans, and a cross section of patients.

Dr. Louis M. Orr, Orlando, Fla., immediate past president of the AMA, has been elected chairman of this new commission. The 14-member commission held an organizational meeting at Miami Beach on June 16.

Resolutions pending before the House of Delegates of the American Medical Association were reviewed by officers of the Texas Medical Association and representatives of Student AMA Chapters at the three Texas medical schools. Dr. J. B. Copeland (second from left), San Antonio, chairman of Texas delegates to the AMA, is shown here reviewing resolutions at the Miami Beach meeting on June 13-17. Looking on are (left to right) William H. Gordon, Jr., Lubbock, Baylor University College of Medicine; Donald R. Payne, Wichita Falls, Southwestern Medical School; Dr. May Owen, Fort Worth, President, Texas Medical Association; and Taylor K. Smith, Houston, University of Texas Medical Branch. For many years, the Texas Medical Association has appropriated funds to help defray expenses for one student from each of the State's three medical schools to attend the annual meeting of the American Medical Association. The students, generally selected by the Student AMA Chapters at their schools, participate in all activities of Texas delegates to the American Medical Association.

Applications Close August 1 For Exhibit Space at AMA Meeting

Applications close on August 1 for scientific exhibit space at the Washington, D. C., clinical meeting of the American Medical Association, scheduled for November 28 to December 1.

Application forms may be received from Charles H. Bramlitt, M.D., Director, Department of Scientific Assembly, American Medical Association, 535 North Dearborn St., Chicago 10.

For the first time at this meeting, the "Hull" award will be presented to the best exhibit on a scientific subject which has not been shown previously at a medical meeting. An honorarium of $250 and a gold medal will be given the winner, and the exhibit will be approved for showing in the scientific exhibit at the 1961 annual meeting of the AMA in New York City.

The award will be presented personally to the recipient by Dr. Thomas G. Hull.

Many Texas Doctors Take Part In AMA Sessions at Miami Beach

Dr. M. O. Rouse, Dallas, has been reelected Vice Speaker of the American Medical Association's House of Delegates for the coming year. Dr. Rouse, who has served in this capacity during the past year, was named unanimously.

During sessions of the AMA House of Delegates, Dr. John K. Glen, Houston, served as a member of the Reference Committee on Legislation and Public Relations. Other Texas delegates who participated in sessions of the AMA House were Dr. J. B. Copeland, San Antonio; Dr. G. W. Cleveland, Austin; Dr. John L. Otto, Galveston; Dr. E. P. Hall, Jr., Fort Worth; Dr. J. C. Terrell, Stephenville; and Dr. T. C. Terrell, Fort Worth.

Dr. Ridings E. Lee, Dallas, has been elected chairman of Alternate Delegates of the American Medical Association. Dr. Lee was elected at a dinner sponsored by AMA Alternate Delegates in Miami Beach. Other Texas physicians who served as alternates at the 1960 annual meeting were Dr. George Turner, El Paso; Dr. Denton Kerr, Houston; Dr. L. Bonham Jones and Dr. John L. Matthews, San Antonio; Dr. J. E. Peavy, Austin; Dr. May Owen, Fort Worth; and Dr. Milton V. Davis, Dallas.

★ **Texas Medical Association**

Plans Near Completion For September 17 Public Relations Conference

Plans are nearing completion for the sixth annual Public Relations Conference, which will be held September 17 at Texas Medical Association headquarters in Austin.

Five authoritative speakers are expected to provide attendants with a wealth of information on public relations. A panel discussion on "What's New in County Medical Society PR" is scheduled for the afternoon session.

The Association's Executive Board will meet at 9:30 a.m. September 18, and many Board, Council, and Committee meetings will be held beginning the evening of September 16. Members of the Woman's Auxiliary are planning their fall Executive Board meeting and a School of Instruction for the 2 days preceding the Conference. Physicians and their wives will be guests of the Association the day of the Conference in the headquarters building. Other entertainment includes a hospitality hour and a football game between the University of Nebraska and the University of Texas the evening of the Conference.

More detailed programs for both Association and Auxiliary meetings will be published in a forthcoming issue of the *Texas State Journal of Medicine.*

Nearly 500 persons attended last year's Conference, which was held in conjunction with an orientation program for new and transfer members of the Association. A similar orientation program will be given during this year's Conference.

Those wishing further information about this fall's meeting may write the Executive Secretary, 1801 North Lamar Boulevard, Austin. Football tickets at $4 each can be ordered from the same office. Hotel reservations should be made by writing directly to the hotel or motor court of choice; physicians should mention that they are members of the Association.

PROFILE IN LEADERSHIP

Virginia-Born Dr. Charles S. Venable Led TMA in Critical War Year 1943

Dr. Charles Scott Venable became seventy-ninth president of the Texas Medical Association during World War II—at one of American Medicine's most challenging periods.

The year was 1943. The annual session first slated for San Antonio, and then for Fort Worth, was never held because of a shortage of doctors to take care of hometown practices, the inadequacy of hotel facilities, and travel restrictions. Only the House of Delegates was able to meet that May in Fort Worth.

When it did meet, there was an understandable pall over House proceedings. All physicians were aware, of course, that upon their shoulders fell much of the responsibility for repairing bodies and minds being ravaged by the worldwide war. One-third of the Texas Medical Association membership was in military service, the rest doing double duty on the civilian front.

The second problem facing Texas doctors at the time of the 1943 House meeting was the recent introduction in Congress of the Wagner-Murray-Dingell bill, which many saw as a major threat to private medicine, more so because it was presented during the Roosevelt administration.

It was in meeting the threat of the socialization of medicine that year that Dr. Venable made his greatest presidential contribution to the Texas Medical Association. Shortly after his installation, he set up a speakers' bureau of doctors to inform the public of Medicine's point of view, and he appointed the Association's first Committee on Public Relations. Under his leadership, the House approved in principle prepayment medical and hospital care plans in the interest of the lower income group.

In a year's time, there was a noticeable shift in the attitude toward socialization of medicine of lay Texans. Dr. Venable's 1944 annual address was entitled "Socialized Medicine Shall Not Pass."

Charles Scott Venable came to Texas in 1907. Then the 30 year old scion of one of Virginia's first families, he arrived in San Antonio on a visit, fell in love with the city, and never returned to Virginia to live.

He was born in Charlottesville, Va., in 1877. His father, Charles Scott Venable, Sr., was a lieutenant colonel on the staff of General Robert E. Lee during the Civil War and a professor of mathematics at the University of Virginia from 1866 to 1896. His mother was the daughter of Valentine W. Southall, speaker of the Virginia Assembly at the time of the state's secession from the Union, and Martha Cocke Southall, a niece of Patrick Henry.

Dr. Venable had received his medical degree from the University of Virginia in 1900 and then gone abroad to do graduate study in London, Paris, Austria, and Dublin. (Just before leaving for Europe, he married his first wife, Madge J. Bonney, who died in 1925. They were the parents of four daughters.) Upon his return, he received an appointment as instructor in the medical department at his alma mater.

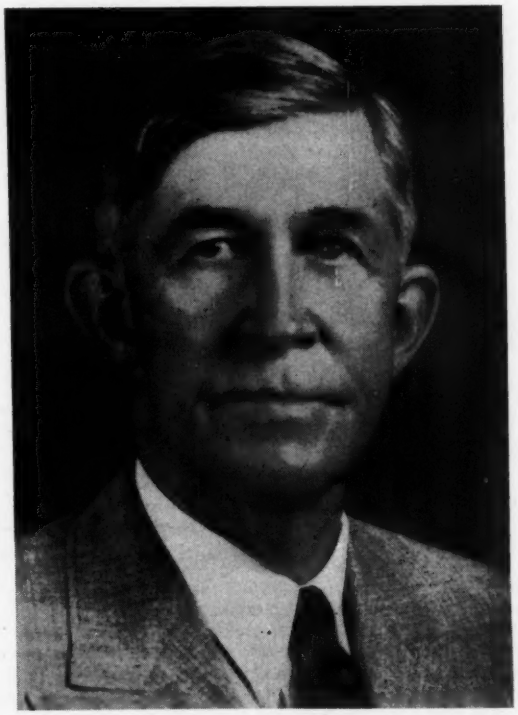
The physician's coming to Texas was to have a distinct impact on San Antonio in several ways. He began his practice there in 1908, specializing in surgery and gynecology. In 1909, he built the Lee Surgical Hospital, which operated for almost 20 years. In 1911, he was instrumental in establishment of the San Antonio Free Clinic, which was the first institution of its kind in southwest Texas.

And, in 1914, he and a group of friends hired the first public health nurse to serve in San Antonio and southwest Texas. Dr. Venable has always claimed that this was accomplished by setting up a "kitty" in a friendly poker game, but actually he and the other members of the group personally underwrote the salary of the nurse.

During World War I, Dr. Venable was co-organizer, with General M. W. Ireland of the Army Medical Corps, of the San Antonio Red Cross chapter, one of the first large, wartime organizations of that sort. He saw overseas service as a medical officer and served as chief of orthopedics in a base hospital, coming home as a lieutenant colonel.

Medicine has been Dr. Venable's principal avocation as well as vocation, his friends report: "When he was not practicing medicine, he was teaching it, writing about it, or doing experimental work. He is one of the many men who have proven beyond doubt that excellent, important research can be done while carrying on an extremely busy practice . . ."

A prolific writer, Dr. Venable has authored some 35 medical articles and was co-author with Dr. Walter G. Stuck of San Antonio of a book: "The Internal Fixation



DR. CHARLES S. VENABLE

of Fractures," published in 1947 in America and Canada. (The authors, both specialists in the field, introduced vitallium, as an especially adaptable alloy, into the field of bone surgery.)

Shortly before he became president of the Texas Medical Association, Dr. Venable developed an adjustable traction splint for use in transportation of patients suffering from fracture of the extremities. Because of the wartime metal shortage, it was made of wood.

While serving as the Association's Fracture Committee chairman, Dr. Venable and his committee prepared a series of articles on first aid for the layman which was published in newspapers in 16 states. He was interested in the treatment of trauma for many years, in this connection serving as a member of the Texas Safety Council and helping to draft the 1943 state law which required ambulances to be equipped with necessary safety and first aid devices and to be served by personnel trained in first aid.

(In a notable explosion in the San Antonio press, Dr. Venable blasted certain "wildcat" Alamo City ambulance services in 1952. "A dead body is worth a darn sight more than somebody brought to a hospital by some of these people," he fumed.)

Medicine rewarded Dr. Venable with many honors through the years. He was 1912 president of the Bexar County Medical Society and Fifth District Councilor from 1912 to 1921. The American Association for the Surgery of Trauma elected him president in 1942. He was one of the founders, in 1922, and later president of the Texas Surgical Society. A fellow of the American College of Surgeons, he has held a number of important positions in that group, and he was a 1941 vice-president of the Southern Surgical Association. For years, he was a consultant in general surgery at Brooke Army Hospital, San Antonio.

When he attended the meetings of the various medical groups to which he belonged, any convention spare time would usually find him in a heated and serious bridge game with friends from all over the world.

Dr. Venable's intense and charming personality won him the friendship and loyalty of many Texas physicians, but he has a certain reputation for frankness among his colleagues. Says one, "Nobody has ever had any difficulty knowing where he stood with Dr. Venable. In his most colorful language, he would soon let you know!"

Today one of the Texas Medical Association's honored emeritus members, Dr. Venable is retired and lives at his San Antonio home with his wife, "Lena." The former Eleanor N. Herff, she is the granddaughter of the late Dr. Ferdinand Herff of San Antonio, who came to Texas in 1849 and was a founder of the Texas Medical Association.

Here, then, another profile in leadership: Dr. Charles Scott Venable, distinguished Texas physician, author, and—defender of private medicine.

TMA's Executive Secretary Honored

At the annual meeting of the Medical Society Executives Association at Miami Beach on June 11, C. Lincoln Williston, Executive Secretary of the Texas Medical Association, was elected to the Board of Directors for a 2 year term.

Mr. Williston has been appointed by the President as chairman of the nominating committee of the group for the coming year.

Donald M. Anderson, Assistant Executive Secretary, Texas Medical Association; Millard J. Heath, Executive Secretary, Dallas County Medical Society; and Roy J. Cates, Executive Secretary, Travis County Medical Society, also attended the MSEA sessions in Miami Beach.

Medical Journal Editor Joins Texas Medical Staff

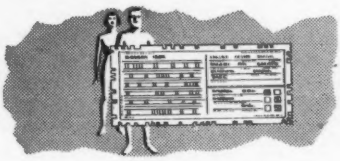
Miss Ruth Trahan, a former editorial assistant of the *Texas State Journal of Medicine* from 1949 to 1953, became managing editor of the Texas Medical Association's *Journal*, July 5.

During Miss Trahan's 4½ year service with TMA, she worked closely with Miss Harriet Cunningham, the late managing editor of the *Journal*, who died May 9 in Austin after working on the *Journal* staff since 1945.

MISS RUTH TRAHAN

Miss Trahan previously was associate editor at the Medical Arts Publishing Foundation in Houston. She also served as assistant editor of *The Heart Bulletin* of the M. D. Anderson Hospital and Tumor Institute in Houston. Miss Trahan served on active duty in the Nurse Corps of the United States Naval Reserve from 1943 to 1946.

Miss Trahan was born and received her primary education in Vinton, La. Prior to receiving her bachelor of journalism degree from the University of Texas in Austin, where she graduated with honors, she was graduated from St. Joseph's School of Nursing in Houston.



★ County Societies

County Medical Society Briefs

The *Nueces County Society* has voted to initiate a $1,000 a year scholarship for a qualified medical student from its area. If a person selected to receive the scholarship continues to merit it, he will get the same amount for all 4 years of his advanced training. To be eligible, the student must enroll in or have completed premedical training and have been accepted by a school of medicine.

To be initiated in the fall, the scholarship program is only the second in the United States sponsored by a county medical society.

Dr. J. R. Riley, Corpus Christi, president of the society, said, "We hope the idea will grow and that many societies will offer similar scholarships."

On June 24, the *Harris County Society* heard Dr. Daniel E. Jenkins, professor of internal medicine at Baylor University College of Medicine, Houston, whose topic was "Widespread Infections Simulating Tuberculosis."

The *Big Bend Society* (Pecos-Jeff Davis-Presidio-Brewster Counties) met June 7, to hear Mr. Elmo Osborne, who outlined the history and program of the American Cancer Society.

The *Gonzales County Society* met at the home of Dr. and Mrs. O. F. von Werssowetz recently. Dr. Walter Reifslager, Austin psychiatrist, made a talk on MAOI drugs for use in general practice.

The *Webb-Zapata-Jim Hogg Counties Society,* in cooperation with the Texas State Department of Health and the H. E. Butt Foundation, has launched a wide-scale pilot study of the incidence of tuberculosis in the area. All school children are to be patch-tested and an extensive mass chest x-ray survey is to be done.

At a recent meeting of the society, Dr. J. T. Lowry spoke on "Narcotics." The meeting was held at Laredo Air Force Base.

The *Tom Green-Coke-Crockett-Concho-Irion-Sterling-Sutton-Schleicher Counties Society* heard Dr. Milton Davis and Dr. Maurice Adams, both of Dallas, present a program on "Congenital and Acquired Heart Disease" at its May 2 session. Slides and a movie indicated surgical procedures.

Date for the next West Texas Tumor Conference was tentatively set for the first Saturday of May, 1961. The group also voted to purchase a projector and flashlight pointer for the society.

"Fluorescence as an Aid in the Cytodiagnosis of Cancer" was the topic of Dr. H. Frank Connally, Jr., and Dr. W. W. Klatt, both of Waco, when the *Hill County Society* met May 13 in Hillsboro. Visitor to the session was Dr. Wendell Lowrey, Gatesville, District 12 representative of the American Cancer Society.

The *Tom Green-Coke-Crockett-Concho-Irion-Sterling-Sutton-Schleicher Counties Society* met in San Angelo April 4, with several members of the American Association of Medical Assistants as special guests.

Two films were shown. The first, presented by the medical assistants group, was "The First Contact." The second, presented by Dr. Eugene C. Winkelmann, San Angelo, dealt with psychotherapeutic drugs and how to control their side effects.

Dr. Tom Hunter was appointed official delegate to the Texas Medical Association, and Dr. Cecil M. French announced plans for an April 24 General Practice Conference in San Angelo. Dr. Sam H. Gainer, San Angelo, gave a report concerning the work of the subcommittee on the Forand Bill.

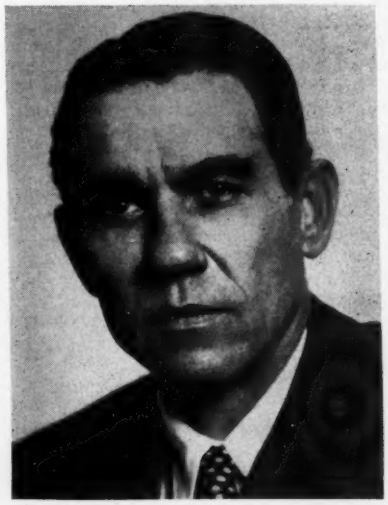
RECENT DEATHS

DR. BRUCE McMILLAN

Dr. Bruce McMillan, Overton, a retired physician, businessman, and civic leader, died April 11, 1960, in a Tyler hospital where he had been under treatment for a respiratory illness for 3 months.

Dr. McMillan was chairman of the board of trustees of the First State Bank of Overton, was managing trustee of the Bruce McMillan, Jr. Foundation, a member of the Kilgore College board of trustees, and he was a charter member and elder of the First Presbyterian Church of Overton.

He had assisted in the establishment of the Overton Memorial Hospital and had served on its board of directors. He had provided numerous scholarships for students at Kilgore College as well as other colleges, and had helped to provide facilities at the Presbyterian Home and Service Agency for Orphans at Itasca. He was responsible for the building of the McMillan Memorial Library in Overton.



DR. BRUCE McMILLAN

An advocate of good forestry practices, Dr. McMillan had operated a ranch near Overton recognized for its cattle raising and high agricultural standards. Shortly before his death he was named an honorary member of the Overton Future Farmers of America. He was awarded posthumously the Distinguished Service Award from the Soil Conservation District, Rusk County, on April 14.

Dr. McMillan and his first wife, the late Mrs. Mary Moore McMillan, had established the McMillan Foundation for their son, Bruce McMillan, Jr., who died July 28, 1951, at the age of 8.

Born April 13, 1904, in Cushing, Dr. McMillan was a graduate of Henderson High School and attended Baylor University at Waco from 1921 to 1923 and the Baylor College of Pharmacy from 1924 to 1925. He was graduated

in 1929 from the medical department of Baylor while it was still in Dallas. From 1930 to 1932 Dr. McMillan served his internship in the Shreveport Charity Hospital, later studying in Vienna.

For four years during World War II, Dr. McMillan served as assistant medical director of the medical department of the Louisiana Ordnance Plant, Shreveport.

A member of the American Medical Association and the Texas Medical Association, Dr. McMillan had belonged to the Smith County Medical Society. He belonged to the Phi Alpha Sigma medical fraternity.

Survivors include his wife, and step-daughter, Catherine Gaile Carroll; his mother, Mrs. O. P. McMillan of San Antonio; two brothers, Dr. Orin McMillan, San Antonio surgeon, and Jess McMillan of Hobbs, N. M.

DR. ROBERT E. PARRISH

Dr. Robert E. Parrish, San Antonio, died May 29, 1960, of coronary thrombosis.

He was born September 11, 1891, in Smithfield, where he received his preliminary education. He attended the University of North Carolina, from 1910 to 1912, receiving his degree on June 6, 1914, from Jefferson Medical College, Philadelphia. He served his internship at Jefferson Medical College Hospital, Philadelphia. In 1916 and 1917 he undertook postgraduate work at the Army Medical School in Washington, D. C.

DR. ROBERT E. PARRISH

He had served 12½ years (from 1916 to 1929) in the United States Army Medical Corps, from which he was discharged as a major.

An eye, ear, nose, and throat specialist, Dr. Parrish was certified by the American Board of Ophthalmology and the American Board of Otolaryngology. He was a fellow of the American Academy of Ophthalmology and Oto-Laryngology.

Besides holding membership in the American Medical Association and the Texas Medical Association, Dr. Parrish had served as president of the Bexar County Medical Society in 1943. He was on the board of directors and a past president of the Robert B. Green Hospital, San Antonio, and he was a civilian consultant at Brooke General Hospital, Fort Sam Houston.

In addition, Dr. Parrish was a senior member of the American Broncho-Esophagological Association and a member of the Texas Otolaryngological Association.

Survivors include his wife of San Antonio; six daughters, Mrs. Helena Ward, Miss Frances Parrish, Mrs. Laura Steinbach, and Mrs. Mary Anderson, all of San Antonio; Mrs. Margaret Liddell, Brownsville; and Mrs. Kathryn McCollum, Dallas; two sons, Robert H. and Charles E., both of San Antonio, and 14 grandchildren.

DR. W. A. KNOLLE

Dr. Waldo Austin Knolle, Brenham, died April 18, 1960, following a heart attack in his office. He had been in conversation with his secretary, Mrs. Grant Woods, when he suffered the attack.

Dr. Knolle, a surgeon, was born in Industry in Austin County on August 15, 1893, the son of Dr. Bernard Knolle and Ida T. (Witte) Knolle.

He attended public school in Industry, Allen Academy at Bryan, and Texas A & M College, College Station. He was a 1917 graduate of Tulane University School of Medicine at New Orleans, where he undertook postgraduate work in 1927 and in 1937.

He married the former Miss Lillie Mae Navratil in 1917 in Brenham, and the couple had two children, Mrs. Dorothy Ann Routt of Chapel Hills, and Marjorie Knolle, who died in 1936 at the age of 17 while a member of the senior class at Brenham High School.

Dr. Knolle was president and chief of staff of the Sarah B. Milroy Hospital at Brenham and of the Washington-Burleson Counties Medical Society. In addition to his county medical membership, he belonged to the Texas Medical Association and the American Medical Association. A member of the Methodist Church, Knights Templar, Shrine, and Masons, Dr. Knolle was also an Elk and a Lion. He was director and honorary trail boss of the Salt Grass Trail, a trail ride from Brenham to Houston to open the Fat Stock Show each year. He also was a director of the Houston Farm and Ranch Club and had been local surgeon

DR. W. A. KNOLLE

of the Santa Fe Railroad for 43 years. He owned the Knolle Hereford Ranch and he also had oil interests.

In 1918, Dr. Knolle served as a first lieutenant in the United States Army.

Survivors include his wife; one daughter, Mrs. Routt; three grandchildren; four sisters, Mrs. A. C. Miller, Carmine; Mrs. Henry Holle, Staten Island, N. Y.; Mrs. L. R. Fordtran, Bellville; and Mrs. Robert Miller, Davis, Calif.; and two brothers, Dr. Roger E. Knolle, Brenham, and Dr. Ben E. Knolle, Rosenberg.

DR. CAROLYN JEANNE LONG

Dr. Carolyn Jeanne Long, Austin pediatrician, died April 7, 1960, in Houston.

Born August 20, 1927, in Waco, Dr. Long was the daughter of H. J. and Eugene (Browne) Long.

She was educated in the public schools of Marshall and was graduated as valedictorian of her class at H. B. Pemberton High School. Later she received the bachelor of science degree from Wiley College, graduating cum laude and as a member of Alpha Kappa Mu Honor Society. She was initiated into Phi chapter of Alpha Kappa Alpha sorority at Wiley College.

DR. CAROLYN JEANNE LONG

Dr. Long received her medical degree from Meharry Medical College, Nashville, in 1950. She served her internship and residency in pediatrics at Homer G. Phillips Hospital, St. Louis, and she was a resident in pediatrics at Harlem Hospital, New York City. She continued her training in Phoenicia, N. Y., and at the University of Texas, Austin.

She began her private practice in Austin, serving on the staffs of Brackenridge and Holy Cross hospitals. In addition, she did research in sickle cell anemia through the University of Texas. At the time of her death, she was active in professional, civic, social, and religious organizations.

She was a member of the American Medical Association, the Texas Medical Association, Lone Star State Medical Association, Travis County Medical Society, Austin Pediatric Society, Travis County Cancer Society, Austin Cerebral Palsy Society, and was on the board of directors of the YWCA. She has contributed articles to medical journals and served on programs at medical assemblies.

Dr. Long was christened in Ebenezer Methodist Church, Marshall, and about a year before her death was confirmed in St. James Episcopal Church, Austin.

Survivors include her mother, Mrs. Eugene B. Long, Tyler; her maternal grandmother, Mrs. Patsy Brown, San Antonio; two aunts, Mrs. Marjorie Brown Hawkins and Mrs. Elaine Aycock, San Antonio; two uncles, Sidney Brown, San Antonio, and F. T. Long, Marshall; and a great uncle, Charles Long, Decatur, Ill.

DR. CARL W. FLEET

Dr. Carl W. Fleet, Houston, died June 4, 1960, in a Houston hospital.

Born March 14, 1912, in Sasakwa, Okla., the general practitioner and surgeon was the son of James Henry and Alice (Brown) Fleet. He received his preliminary education at Ada, Okla. He took his bachelor of science degree in 1932 from Oklahoma University, Norman, and his medical degree in 1937 from Baylor University College of Medicine while it was still located in Dallas. He also attended the University of California at Los Angeles.

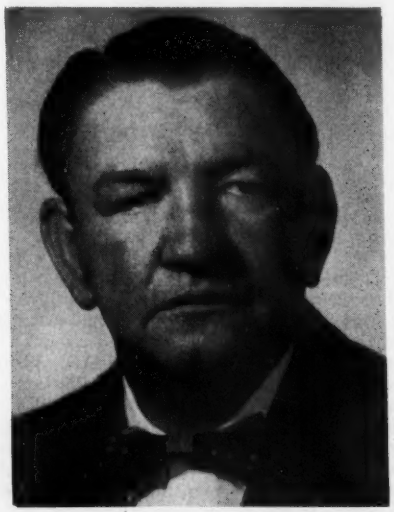
Dr. Fleet served his internship at Kansas City General Hospital, Kansas City, Mo., and his residency at Methodist Hospital in Houston.

Married November 11, 1933, at Dallas to Miss Totts Foster, Dr. Fleet and his wife became the parents of one daughter, Miss Alice Jo Fleet, now Mrs. A. J. Dickey of Dallas.

A member of the American Medical Association, the Texas Medical Association, and the Harris County Medical Society, Dr. Fleet also belonged to the Houston Club, the Elks, and the Baptist Church.

From 1941 to 1946, he served with the United States Army Medical Corps.

Survivors include his wife, his daughter, one grandson, and a brother, John J. Fleet of Ada, Okla.



DR. CARL W. FLEET

MEMBERSHIP

TEXAS MEDICAL ASSOCIATION

JULY, 1960

The membership list which follows is compiled from names sent by county society secretaries to the state office. The names are listed by county societies within the fifteen districts. The number after each county listed below indicates the district in which the county is located.

Anderson—11
Andrews—2
Angelina—10
Aransas—6
Archer—13
Armstrong—3
Atascosa—5
Austin—9
Bailey—3
Bandera—5
Bastrop—7
Baylor—13
Bee—6
Bell—12
Bexar—5
Blanco—7
Borden—2
Bosque—12
Bowie—15
Brazoria—8
Brazos—12
Brewster—1
Briscoe—3
Brooks—6
Brown—4
Burleson—9
Burnet—7
Caldwell—7
Calhoun—8
Callahan—13
Cameron—6
Camp—15
Carson—3
Cass—15
Castro—3
Chambers—10
Cherokee—11
Childress—3
Clay—13
Cochran—3
Coke—4
Coleman—4
Collin—14
Collingsworth—3
Colorado—8
Comal—5
Comanche—4
Concho—4
Cooke—14
Coryell—12
Cottle—3
Crane—4
Crockett—4
Crosby—3
Culberson—1
Dallam—3
Dallas—14
Dawson—2
Deaf Smith—3
Delta—14
Denton—14
De Witt—8
Dickens—2
Dimmit—5
Donley—3
Duval—6
Eastland—13
Ector—2
Edwards—5
Ellis—14
El Paso—1
Erath—12
Falls—12
Fannin—14
Fayette—8
Fisher—2
Floyd—3
Foard—3
Fort Bend—8
Franklin—14
Freestone—11
Frio—5
Gaines—2
Galveston—8
Garza—2
Gillespie—5
Glasscock—2
Goliad—8
Gonzales—5
Gray—3
Grayson—14
Gregg—15
Grimes—9
Guadalupe—5
Hale—3
Hall—3
Hamilton—12
Hansford—3
Hardeman—3
Hardin—10
Harris—9
Harrison—15
Hartley—3
Haskell—13
Hays—7
Hemphill—3
Henderson—11
Hidalgo—6
Hill—12
Hockley—3
Hood—12
Hopkins—14
Houston—11
Howard—2
Hudspeth—1
Hunt—14
Hutchinson—3
Irion—4
Jack—13
Jackson—8
Jasper—10
Jeff Davis—1
Jefferson—10
Jim Hogg—6
Jim Wells—6
Johnson—12
Jones—13
Karnes—5
Kaufman—14
Kendall—5
Kenedy—6
Kent—2
Kerr—5
Kimble—4
King—2
Kinney—5
Kleberg—6
Knox—13
Lamar—14
Lamb—3
Lampasas—7
LaSalle—5
Lavaca—8
Lee—7
Leon—11
Liberty—10
Limestone—12
Lipscomb—3
Live Oak—6
Llano—7
Loving—1
Lubbock—3
Lynn—2
McCulloch—4
McLennan—12
McMullen—6
Madison—9
Marion—15
Martin—2
Mason—4
Matagorda—8
Maverick—5
Medina—5
Menard—4
Midland—2
Milam—12
Mills—4
Mitchell—2
Montague—13
Montgomery—9
Moore—3
Morris—15
Motley—3
Nacogdoches—10
Navarro—12
Newton—10
Nolan—2
Nueces—6
Ochiltree—3
Oldham—3
Orange—10
Palo Pinto—13
Panola—11
Parker—13
Parmer—3
Pecos—1
Polk—9
Potter—3
Presidio—1
Rains—14
Randall—3
Reagan—4
Real—5
Red River—15
Reeves—1
Refugio—6
Roberts—3
Robertson—12
Rockwall—14
Runnels—4
Rusk—11
Sabine—10
San Augustine—10
San Jacinto—9
San Patricio—6
San Saba—4
Schleicher—4
Scurry—2
Shackelford—13
Shelby—10
Sherman—3
Smith—11
Somervell—12
Starr—6
Stephens—13
Sterling—4
Stonewall—2
Sutton—4
Swisher—3
Tarrant—13
Taylor—13
Terrell—5
Terry—2
Throckmorton—13
Titus—15
Tom Green—4
Travis—7
Trinity—9
Tyler—10
Upshur—15
Upton—4
Uvalde—5
Val Verde—5
Van Zandt—14
Victoria—8
Walker—9
Waller—9
Ward—1
Washington—9
Webb—6
Wharton—8
Wheeler—3
Wichita—13
Wilbarger—13
Willacy—6
Williamson—7
Wilson—5
Winkler—1
Wise—13
Wood—11
Yoakum—2
Young—13
Zapata—6
Zavala—5

(Int.) indicates Intern Membership. (Hon.) indicates Honorary Membership. (Emer.) indicates Membership Emeritus. (Inac.) indicates Inactive Membership. (Mil.) indicates Military Membership.

FIRST DISTRICT

Dr. C. E. Oswalt, Jr., Fort Stockton, Councilor.

EL PASO

Anderson, Helen W., 1501 Arizona, El Paso.
Appel, Saul B., 1501 Arizona, 10E, El Paso.
Arguelles, F. L., (Hon.),
401 S. Stanton, El Paso.
Autrey, Walter Claude,
8030 N. Loop Dr., El Paso.
Avner, S. Leighton, 1900 N. Oregon, El Paso.
Awe, Chester D., 1501 Arizona St., El Paso.
Ayub, Pablo, 4622 Alameda Ave., El Paso.
Baca, Francisco E., 624 Caples Bldg., El Paso.
Barrett, Frank O., (Hon.),
1501 Arizona St., El Paso.
Basom, W. Compere,
1220 N. Stanton St., El Paso.
Bell, Herbert J., 3920 Idalia, El Paso.
Bennett, J. Travis, 1501 Arizona St., El Paso.
Bennett, Raymond J.,
1501 Arizona St., El Paso.
Bernard, Jack A., 1501 Arizona St., El Paso.
Bernell, Edward C.,
1017 1st Natl. Bldg., El Paso.
Black, Arthur P., 525 Montana St., El Paso.
Black, Gordon L., 1501 Arizona St., El Paso.
Boehler, Clement C., 1501 Arizona St., El Paso.
Bornstein, Frederick P.,
Providence Mem. Hosp., El Paso.
Boverie, Robert F.,
415 E. Yandell Blvd., El Paso.
Bozzell, James D., 1501 Arizona St., El Paso.
Breck, Louis W., 1220 N. Stanton St., El Paso.
Britton, Bloyce H., 505 1st Natl. Bldg., El Paso.
Brunner, George, 1208 Mills Bldg., El Paso.
Budwig, Ira A., 1501 Arizona St., El Paso.
Byrne, Basil K., 1501 Arizona St., El Paso.
Cameron, David M., 1501 Arizona St., El Paso.
Cardwell, Robert J., 1900 N. Oregon, El Paso.
Carnes, David M., 125 Snelson Dr., Ysleta.
Carpenter, Gray E., 2323 Montana St., El Paso.
Carter, Joe C., 214 Banner Bldg., El Paso.
Casavantes, Luis, 914 N. Stanton St., El Paso.
Causey, Oscar M., 2927 Montana, El Paso.
Caylor, Robert N., 1900 N. Oregon, El Paso.
Clayton, Ralph S.,
415 E. Yandell Blvd., El Paso.
Coldwell, William I.,
1900 N. Oregon St., El Paso.
Cooper, Arlin B., 1501 Arizona St., El Paso.
Craige, Branch, 2432 Savannah, El Paso.
Crossett, Egbert Samuel,
1501 Arizona, El Paso.
Cummins, Erwin J., 1015 Mills Bldg., El Paso.
Curtis, Wickliffe R., 1501 Arizona St., El Paso.
Damiani, Ann B., 1900 N. Oregon, El Paso.
Davidson, Maurice C.,
1501 Arizona St., El Paso.
Davis, George R., Box 1524, El Paso.
Davis, W. J., (Hon.),
Route 1, Box 80-A, Anthony, N. M.
Del Campo, Dante, 5737 Trowbridge, El Paso.
Demarest, Harry W., 5532 Bandy Rd., El Paso.
Deter, Russell L., 1501 Arizona St., El Paso.
Dietrich, Hervey W., 415 E. Yandell, El Paso.
Domenicali, Pete, Jr.,
7705 N. Loop Rd., El Paso.
Don, Rita Louisa, 1900 N. Oregon, El Paso.
Dow, Antonio, 1900 N. Oregon, El Paso.
Eck, Andrew J., 5970 Alameda Ave., El Paso.
Edwards, George M., (Hon.),
2507 Nasworthy Dr., San Angelo.
Egbert, O. Edward, Jr.,
2526 Frankfort Ave., El Paso.
Egbert, Orville E., 1501 Arizona St., El Paso.
Ellis, Jack Reese, 1501 Arizona St., El Paso.
Elsberg, Charles P., 1900 N. Oregon, El Paso.
Emmett, John E., 1900 N. Oregon, El Paso.
Engel, Eugen, 329 E. San Antonio, El Paso.
Epstein, I. M., 525 Montana, El Paso.
Evans, Fred G., 1900 N. Oregon, El Paso.
Evans, Ward, 1900 N. Oregon, El Paso.
Ewalt, Donald H., 1501 Arizona St., El Paso.
Feener, Lester C., 404 Banner Bldg., El Paso.
Fehlman, Bernard F., Jr.,
1501 Arizona, El Paso.
Feinberg, Walter D.,
1501 Arizona, 14D, El Paso.
Fernandez, Carlos A.,
5980 Alameda Ave., El Paso.
Floyd, Joe R., 1501 Arizona St., El Paso.
Frerichs, John B., 1900 N. Oregon, El Paso.
Gaddis, Leo R., 1223 Mills Bldg., El Paso.
Gaddis, William R., 1223 Mills Bldg., El Paso.
Gaddy, S. J., 912 N. Mesa Ave., El Paso.
Galatzan, Joe S., 2911 Pershing Dr., El Paso.
Gallagher, Paul, (Hon.),
1423 Del Rio Blvd., Eagle Pass.
Gardea, Raymond A.,
4618 Alameda Ave., El Paso.
Garrett, Henry D., 1501 Arizona St., El Paso.
Gemoets, Albert A., 3726½ Alameda, El Paso.
Gibson, H. M., Jr.,
1900 N. Oregon St., El Paso.
Gilmore, Richard C., Jr.,
927 Raynolds Blvd., El Paso.
Gladstone, Larry A.,
1501 Arizona, 14D, El Paso.
Goldfarb, Irvin J., 1501 Arizona, El Paso.
Golding, Frank C., 1501 Arizona St., El Paso.
Gonzalez, Saul, (Int.),
Hotel Dieu Hosp., El Paso.
Goodloe, B. Lynn, 415 E. Yandell, El Paso.
Gorman, James J., 701 1st Natl. Bldg., El Paso.
Grass, Adrian L., 1220 N. Stanton, El Paso.
Green, Charles L.,
105 Med. Arts Bldg., El Paso.
Green, J. Leighton, 1501 Arizona St., El Paso.
Hardie, Bradford, 403 1st Natl. Bldg., El Paso.
Hart, Maynard S., 1501 Arizona St., El Paso.
Hassler, Bert G., (Mil.),
139 U. S. Ct. House, El Paso.
Hatfield, Haskell D., 1501 Arizona St., El Paso.
Heller, Solomon, 1900 N. Oregon, El Paso.
Higdon, Donald A., Box 938, Fabens.
Hinton, Joseph H., 1900 N. Oregon, El Paso.
Holt, Russell, 415 E. Yandell, El Paso.
Homan, Ralph H., 1501 Arizona St., El Paso.
Homan, Robert B., Jr.,
1501 Arizona St., El Paso.
Hornedo, Manuel D.,
118 W. Missouri St., El Paso.
Hornisher, J. J., 4521 Leeds, El Paso.
Hunter, C. D., 800 Montana St., El Paso.
Hunter, Robert C., Jr., (Mil.),
WBAH, El Paso.

Iwen, George W., 1501 Arizona, El Paso.
Jabalie, Edward N., 125 Snelson Dr., Ysleta.
Jenness, Burt F., 3418 Fort Blvd., El Paso.
Johnstone, John H., 125 Snelson Dr., Ysleta.
Jones, W. A., 1501 Arizona St., El Paso.
Jordan, Gerald H., 1501 Arizona St., El Paso.
Kearney, William W., (Inac.), Hawkeye, Iowa.
Keller, Nathan H., El Paso Natl. Bldg., El Paso.
King, Sam R., (Inac.),
5020 Vista Del Monte, El Paso.
Kleban, M. Nathan,
1900 N. Oregon St., El Paso.
Kosicki, Zigmund W.,
1220 N. Stanton St., El Paso.
Kurita, Kenneth S., 706 Caples Bldg., El Paso.
Landis, Gilbert, 1501 Arizona, 3C, El Paso.
Leigh, Harry, (Inac.),
2619 Altura Ave., El Paso.
Leonard, Morton H.,
1220 N. Stanton St., El Paso.
Licon, Francisco, 3127 Alameda Ave., El Paso.
Liddell, Thos. C., 609 1st Natl. Bldg., El Paso.
Lipsey, Billy C., Box 487, Van Horn.
Logsdon, Charles P. C.,
Medical Arts Bldg., El Paso.
Lombard, Julian H., 4620 Emory Way, El Paso.
Lorentzen, Wayne L., 1900 N. Oregon, El Paso.
Lozano, Porfirio, 417 S. Stanton, El Paso.
Luckett, Alfred E., 1501 Arizona St., El Paso.
Lukowski, John L., 3313 Fort Blvd., El Paso.
Marshall, Howard J. H.,
1501 Arizona St., El Paso.
Martin, John D., 1501 Arizona, 14, El Paso.
Martin, T. Sterling, 1501 Arizona St., El Paso.
McGuire, John M., 105 Isabella, El Paso.
McNeil, James Lewis,
1501 Arizona St., El Paso.
McVaugh, Charles C., 5015 Montoya, El Paso.
Melgar, Alberto, 4622 Alameda Ave., El Paso.
Miller, John B., 3313 Fort Blvd., El Paso.
Molinar y Rey, Jose, (Inac.),
1106 Mundy Ave., El Paso.
Molinar Z, Ramon, 306 S. Stanton, El Paso.
Molloy, Maxwell S., 415 E. Yandell, El Paso.
Morgan, James R., 1501 Arizona, 3A, El Paso.
Morrison, John E., 1011 Mills Bldg., El Paso.
Morrow, Walter G., Jr.,
1900 N. Oregon, El Paso.
Multhauf, A. W., 1515 1st Natl. Bldg., El Paso.
Mutnick, Reuben, 112 S. Concepcion, El Paso.
Nering, A. Robert,
1025 1st Natl. Bldg., El Paso.
Palafox, Mario, 1220 N. Stanton St., El Paso.
Pastrana, Raul, 411 Caples Bldg., El Paso.
Perry, Alvin L., 209 Med. Arts Bldg., El Paso.
Persky, Murray, 1501 Arizona, 15B, El Paso.
Peticolas, John D., 318 Mills Bldg., El Paso.
Phillips, Richard J., (Mil.),
U. S. AEHL, Army Chem. Cen., Md.
Pierce, Wendell L., 415 E. Yandell, El Paso.
Ponsford, John A., 1501 Arizona St., El Paso.
Postlewaite, Jack C., 1501 Arizona St., El Paso.
Price, Elwyn D., (Inac.),
1013 Mills Bldg., El Paso.
Prieto, Philip M., 1501 Arizona St., El Paso.
Quirarte, Humberto, 415 E. Yandell, El Paso.
Race, Charles G., Jr., 1501 Arizona, El Paso.
Ravel, Vincent M., 1900 N. Oregon, El Paso.
Redelfs, John Wright,
1501 Arizona St., El Paso.
Rennick, Charles F., 4802 Montana, El Paso.
Reynolds, Chester Lee,
3016 Wyoming St., El Paso.
Rheinheimer, E. W., 415 E. Yandell, El Paso.
Rice, Herman, 1501 Arizona St., El Paso.
Rigney, Paul, 821 E. Yandell Blvd., El Paso.
Rissler, Ross W., 2001 Grant Ave., El Paso.
Robbins, Jacob B., 1900 N. Oregon, El Paso.
Rodarte, Ruben B., 401 S. Stanton, El Paso.
Rodriguez, Aurora, 504 S. Stanton St., El Paso.
Rogde, Jacob, 315 Mills Bldg., El Paso.
Rogers, S. Perry, 1501 Arizona St., El Paso.
Roman, Jose, Jr., 4622 Alameda Ave., El Paso.
Schlenker, George M.,
1900 N. Oregon, El Paso.
Schuessler, Willard W.,
1501 Arizona St., El Paso.
Schuster, Frank P., 1501 Arizona, El Paso.
Schuster, Frank P., Jr.,
1900 N. Oregon, El Paso.
Schuster, Stephen A., 1501 Arizona, El Paso.
Shugart, Joseph A., 1501 Arizona St., El Paso.
Skemp-Nystrom, Harriet, Box 25, Canutillo.
Smith, Leslie M., 1501 Arizona St., El Paso.
Smith, William G., 415 E. Yandell, El Paso.
Snyder, Richard D., (Mil.),
23 Merritt Ave., Dumont, New Jersey.
Soloff, Philip N., 1900 N. Oregon, El Paso.
Sorensen, Alfred, 600 Loretto Rd., El Paso.
Soto, Raul C., 522 Caples Bldg., El Paso.
Spearman, Maurice P.,
1501 Arizona St., El Paso.
Spier, Erich, 415 E. Yandell Blvd., El Paso.
Stanfill, Chas. M., 1900 N. Oregon, El Paso.
Stapp, Celso C., 1900 N. Oregon, El Paso.
Stern, J. Edward, 1900 N. Oregon, El Paso.
Stevens, B. F., (Hon.), 217 Blacker, El Paso.
Stowe, Jesson L., 2323 Montana St., El Paso.
Stratemeyer, W. P., 1501 Arizona, El Paso.
Taber, Ben Zion, 415 E. Yandell, El Paso.
Thayer, Robert H., 1501 Arizona St., El Paso.
Thomas, Merle D., 1501 Arizona St., El Paso.
Thompson, Robert F.,
1900 N. Oregon, El Paso.
Treece, Angus A., Fabens.
Trowbridge, James O., (Mil.),
Fed. Correctional Inst., La Tuna.
Tubbs, William M., 2929 Montana, El Paso.
Turner, George, 1501 Arizona St., El Paso.
Unger, Albert H., 911 Huckleberry St., El Paso.
Vandevere, William E.,
1900 N. Oregon, El Paso.
Vargas G, Francisco, 408 S. Stanton, El Paso.
Varner, Harry H., 1501 Arizona, 5 E, El Paso.
Vaughan, W. Hunter,
1501 Arizona St., 2B, El Paso.
Verosky, John M., 1501 Arizona St., El Paso.
Villareal, Leopoldo, Caples Bldg., El Paso.
Vinikoff, Maurice R.,
1218 Mills Bldg., El Paso.
Voigt, Alfred E., 4806 Montana St., El Paso.
Von Briesen, Delphin,
1501 Arizona St., El Paso.
Walker, Jack A., Jr., 1501 Arizona St., El Paso.
Walker, Newton F.,
403 1st Natl. Bldg., El Paso.
Webb, Charles E., 1501 Arizona St., El Paso.
White, John E., 1501 Arizona, 2A, El Paso.
Wilcox, Leigh E., 1501 Arizona St., El Paso.
Wollmann, Walter W.,
2001 Grant Ave., El Paso.
Young, John J., 800 Montana St., El Paso.
Zacharias, Otis G., 606 Loretto Rd., El Paso.

PECOS-JEFF DAVIS-PRESIDIO-BREWSTER

Blackwell, James H., (Hon.), Marfa.
Blow, Frank T., (Hon.), Alpine
Gaddis, Don A., Fort Davis.
Gipson, James F.,
211 N. Nelson, Fort Stockton.
Hill, Malone V., 123 N. Sixth, Alpine.
Hoffman, George A.,
106 S. Main, Fort Stockton.
Hundley, John C., Box 466, Fort Stockton.
Jones, Charles E., Box 367, Fort Stockton.
Kelley, William N., (Hon.),
108 Sue St., Crane.
Lancaster, James D., Box 277, Fort Stockton.
Lockhart, William E., Jr., 401 N. 4th, Alpine.
O'Donnell, John W., Box 993, Alpine.
Oswalt, Chas. E., Jr.,
201 N. Water St., Fort Stockton.
Pate, John W., 202 N. Second, Alpine.
Ponton, Arvel R., Jr., 202 N. 2nd St., Alpine.
Richardson, Gene A., Box 84, Sanderson.
Searls, John P., Marfa.
Sherrod, Vincent A., Box 608, Iraan.
Sibley, D. J., Jr., Box 367, Fort Stockton.
Stover, Walter H., 112 E. Texas, Marfa.
Wright, Joel E., (Hon.), Box 697, Alpine.

REEVES-WARD-WINKLER-LOVING-CULBERSON-HUDSPETH

Avery, Harlow F., Trans-Pecos Clin., Pecos.
Barnett, Arthur J., 310 S. Gary, Monahans.
Camp, Jim, Box 899, Pecos.
Campbell, Charles F., 111 E. Halley, Kermit.
Dampeer, John O., Jr., Kermit.
Denney, Ernest E., 909 S. Robinson, Kermit.
Dowling, George W.,
Winkler Co. Hosp., Kermit.
Dunn, John P., 8th & Elm, Pecos.
Garrett, Donald C., 310 S. Gary, Monahans.
Gibson, Joseph V., Jr.,
812 Meyer Lane, Kermit.
Hay, Bruce H. H., 401 Ross Blvd., Pecos.
Heath, Joe D., 113 S. Mulberry, Kermit.
Kunstadt, Paul, Monahans.
Lindley, Harold, Pecos.
McClure, Wayne H., Kermit.
Munk, Otto, Monahans.
Peddicord, Harper, II, 113 W. Halley, Kermit.
Peddicord, Orene W., 113 W. Halley, Kermit.
Prout, Fred J., Monahans.
Reedy, Jack D., 1703 W. Fourth, Pecos.
Rehmeyer, Walter O., Monahans.
Roberts, Rufus A., Box 747, Pecos.
Robinson, Cecil A., Box 487, Kermit.
Robinson, Lila Rose, Box 487, Kermit.
Schmidt, Ed W., Box 1290, Pecos.

SECOND DISTRICT

Dr. Henrie E. Mast, Midland, Councilor

ANDREWS-ECTOR-MIDLAND

Autry, Paul G., 2010 W. Illinois, Midland.
Barganier, John H., 601 W. Fourth St., Odessa.
Bauman, John E.,
413 N. Lincoln Ave., Odessa.
Blonkvist, Brent G., 2005 W. Wall, Midland.
Bobo, Thomas C., 805 W. Wall, Midland.
Boone, Martin H., Jr.,
210 North C St., Midland.
Brantly, E. Clayton, Jr.,
1207 E. Tenth St., Odessa.
Bryan, John N., 1200 N. Texas St., Odessa.
Buck, Roger, 304 North N, Midland.
Bugg, Robert N., 208 N. Garfield, Midland.
Cantrell, David E., 516 W. 4th St., Odessa.
Carson, Willis T., 506 N. Alleghaney, Odessa.
Casey, James G., Box 457, Andrews.
Chapple, James H., (Inac.),
P. O. Box 5366, Midland.
Clader, Durwood N., 2310 W. Ohio, Midland.
Clark, John L., Jr., 1515 N. Texas, Odessa.
Coleman, Jesse L., 210 North C St., Midland.
Collins, Reed, 2310 W. Ohio, Midland.
Cone, Jesse D., 318 N. Alleghaney, Odessa.
Connery, David B., 2006 W. Ohio, Midland.
Curry, Dale W., 1200 N. Texas, Odessa.
Darwin, Lloyd K.,
1600 N. Texas Ave., Odessa.
Daves, Elizabeth A.,
505 N. Alleghaney, Odessa.
Daves, James A., 505 N. Alleghaney, Odessa.
Dempsey, Edwin B., P. O. Box 1987, Andrews.
Driscoll, Edward T.,
2010 W. Illinois, Midland.
Edmondson, Paul J., 1147 E. 42nd St., Odessa.
Elliott, Thomas J., Box 1631, Odessa.
Elliott, Vance J., Medical Arts Clin., Odessa.
Finch, Albert B., 1200 N. Texas Ave., Odessa.
Foster, John W., 1803 W. Wall St., Midland.
Fulcher, Oliver A., 520 W. 4th St., Odessa.
Furst, William D., 1209 E. 10th St., Odessa.
Gaarde, Fred W., 501 Andrews Hwy., Midland.
Gomillion, Jesse D., 1802 W. Wall, Midland.
Gooch, J. Oliver, Medical Arts Bldg., Midland.
Grafa, Barney G., Jr., 1806 W. Wall, Midland.
Grammer, John C., 1802 W. Wall, Midland.
Green, Wilbur K., 404 N. Washington, Odessa.
Greenlee, Ralph G., 401 N. Garfield, Midland.
Greenlees, David L., 518 W. 4th St., Odessa.
Grice, Marvin Edward,
313D N. Alleghaney, Odessa.
Guerriero, Chas. P., III,
2009 W. Wall St., Midland.
Hays, Alan L., Headlee Clinic, Odessa.
Hays, Johnnie W.,
314 N.W. Avenue B, Andrews.
Headlee, Emmet V., Box 3112, Odessa.
Hestand, Haskell E., 421 N. Golder, Odessa.
Hilliard, Joe B., 401 N. Garfield, Midland.
Hines, J. Clarence,
6715 Richmond Rd., Houston.
Horne, Albert M., 2200 W. Illinois, Midland.
Horton, George W., 513 W. 4th, Odessa.
Hubbard, Prevost, Jr., 517 W. 4th St., Odessa.
Huffman, Phyllis J., 409 N. Garfield, Midland.
Hunsaker, R. Dale, 2203 W. Illinois, Midland.
Hunt, H. Lynn, Jr., 421 N. Golder, Odessa.
Hunt, Jackie H., 408 N. Washington, Odessa.
Hutcheson, Zenas W., Jr.,
P. O. Box 1987, Andrews.
Johnson, Chester R., 2009 W. Wall, Midland.
Johnson, George L., Jr.,
500 N. Alleghaney, Odessa.
Johnson, Homer B.,
501 Andrews Hwy., Midland.
Johnson, Reinhold E.,
3318 W. Michigan Ave., Midland.
Kirk, Earl H., 314 N. Alleghaney, Odessa.
Klempnauer, Richard G.,
2310 W. Ohio, Midland.
Lang, Garland H., 2009 W. Wall, Midland.
Leigh, Henry T., Jr., 206 N.E. First, Andrews.
Lekisch, Kurt, Box 852, Midland.
Loftis, J. Stewart, 1165 E. 42nd, Odessa.
Loring, Milton J., 2001 W. Wall St., Midland.
Lunn, William W., 408 N. Alleghaney, Odessa.
Madsen, Martha E., 2200 W. Illinois, Midland.
Marinis, Thomas P., Box 4966, Midland.
Mason, Stuart A., 1147 E. 42nd St., Odessa.
Mast, Clarence S., 2203 W. Illinois, Midland.
Mast, Henrie E., 2203 W. Illinois, Midland.
Mast, John R., 2203 W. Illinois, Midland.
McAlister, Joseph H.,
313B N. Alleghaney, Odessa.
McCullough, Edison W.,
1415 N. Big Spring, Midland.
McIntire, Thornton S., 515 W. 4th St., Odessa.
Melton, Thos. June, Jr.,
501 Andrews Hwy., Midland.
Melton, William H.,
Med. Center Hosp., Odessa.

Mickle, Edwin R., 2011 West Ohio, Midland.
Mitis, Charles Z. K.,
313D N. Alleghaney, Odessa.
Mood, George F., 2203 W. Illinois, Midland.
Nichols, Myron McCall,
Univ. of Tex. Med. Br., Galveston.
Norton, Richard G., Box 4673, Midland.
Novak, Theodore W., 413 N. Lincoln, Odessa.
Novosad, Charles L., Jr.,
400 E. 19th St., Odessa.
Oehlschlager, F. Keith,
1167 E. 42nd St., Odessa.
Orr, Marjorie K., 206 North O, Midland.
Page, Edwin C., 513 W. 4th, Odessa.
Parks, Walter S., Jr., 2009 W. Wall, Midland.
Payne, William T., 408 N. Alleghaney, Odessa.
Peer, George F., 413 N. Lincoln, Odessa.
Penn, Rhesa L., Jr., Box 5073, Midland.
Rader, J. Paul, 313 N. Alleghaney, Odessa.
Rainer, James W., (dead), Odessa.
Ramsey, Richard R., P. O. Box 1987, Andrews.
Shapira, Jake, 501 Andrews Hwy., Midland.
Sheets, John H., 1155 E. 42nd St., Odessa.
Simpson, Fredric E.,
410 N. Washington, Odessa.
Stephens, Charles A., 1509 N. Texas, Odessa.
Storey, Wray D., Box 1631, Odessa.
Tannenholz, Harold S., 1149 E. 42nd, Odessa.
Thornton, Elbert H. E., 500 N. Dotsy, Odessa.
Tompkins, Harry H., Jr.,
P. O. Box 1987, Andrews.
Turner, Jack Lee, 601 W. 4th, Odessa.
Van Leeuwen, Gerard,
410 N. Washington, Odessa.
Walker, H. Glenn, (Int.),
3206 Albans, Houston.
Walton, Jack R., 2011 W. Ohio, Midland.
White, Robert N., 210 North C, Midland.
Wiesner, William A.,
406 N. Washington, Odessa.
Williams, Glenn R., 413 N. Lincoln, Odessa.
Wood, Barry P., 4009 Milton, Houston.
Wood, John K., 601 W. 4th St., Odessa.
Wright, Robert L., 601 W. Fourth, Odessa.
Wyvell, Dorothy B., 307 North M, Midland.
Youngblood, Billy J., 1802 W. Wall, Midland.
Zang, Louis C., 1219 E. 10th, Odessa.
Zinterhofer, Louis J., Box 71C, Rt. 2, Midland.

BORDEN-DICKENS-GARZA-KENT-KING-SCURRY-STONEWALL

Alexander, Arthur B., Box 1092, Spur.
Cockrell, C. Ray, 2612 Avenue R, Snyder.
Dillaha, Carl A., Jr., P. O. Box 1086, Snyder.
Hartley, Thomas F., P. O. Box 1086, Snyder.
Jones, Wilton N., 2601 Avenue F, Snyder.
Martin, Edward H., (Int.),
218 Cascade Rd., Springfield, Pa.
Moore, Ben E., P. O. Box 1086, Snyder.
O'Banion, John Wm., Jr.,
Med. Arts Bldg., Snyder.
Pierce, Robert B., 1903 37th St., Snyder.
Raines, John W., Snyder Hosp. Clin., Snyder.
Redwine, Harry P., Box 295, Snyder.
Rogers, Edward A., Jr.,
Medical Arts Bldg., Snyder.
Ward, Harry W., Route 3, Box 21 A, Snyder.

DAWSON-LYNN-TERRY-GAINES-YOAKUM

Bailes, Jerry Ray, 602 West Tate, Brownfield.
Barnes, Louis R., Jr.,
Seagraves Clinic, Seagraves.
Black, Douglas B., Box 90, Lamesa.
Daniell, Alfred H., 919 E. Main, Brownfield.
Dow, Harold D., Box 546, Seminole.
Frazier, Sam Z., Lamesa.
Greenfield, Keller P., Box 1096, Denver City.
Hill, Wayne C., Brownfield.
Hood, Steve E., Jr.,
Gaines Clin. Hosp., Seminole.
Knox, Cecil B., Jr., Box 1026, Brownfield.
Koberg, Frederick J., Seminole.
Lehman, Joe M., (Mil.),
2113 South S St., Fort Smith, Ark.
McKay, J. Vernon, 613 S. 1st St., Lamesa.
Powers, Ace G., Seagraves Cl. Hosp., Seagraves.
Price, Noble H., Lamesa.
Prohl, Emil H., Tahoka.
Puckett, John P., Lamesa.
Rising, Ernest E., Jr.,
207 S. 6th St., Brownfield.
Rumbo, Noble L., Box D, O'Donnell.
Seale, Francis E., 1008 N. 3rd St., Lamesa.
Sharp, Joseph E., Denver City.
Smith, Alfred H., (Hon.), Lamesa.
Staker, Norman W., Lamesa.
Stone, Noah W., 207 S. 6th St., Brownfield.
Thomas, C. Skiles, Tahoka.
Tinley, Robert E., Denver City.
Treadaway, Thomas L.,
601 E. Tate, Brownfield.
Watts, Maurice A., Denver City.

HOWARD-MARTIN-GLASSCOCK

Broadrick, Broadway, Drawer 949, Big Spring.
Brohn, Alfred J., P. O. Box 882, Big Spring.
Burnett, Jack H., Jr., 811 Main St., Big Spring.
Carson, Arch D., 1500 Gregg, Big Spring.
Childs, James L., Box 231, Big Spring.
Cowper, Roscoe B. G., Box 111, Big Spring.
Dillon, George F., 811 Main St., Big Spring.
Fish, John H., Box 1191, Big Spring.
Friedewald, Vincent E.,
811 Main St., Big Spring.
Hall, Granville T.,
P. O. Box 1191, Big Spring.
Harrison, Preston E.,
Big Spring State Hosp., Big Spring.
Hogan, John E., Box 991, Big Spring.
Lurting, Frederick W.,
811 Main St., Big Spring.
Malone, Phocian W., Box 991, Big Spring.
Marcum, Carlo B., 811 Main St., Big Spring.
Mays, Floyd R., Jr., Box 191, Big Spring.
Peacock, George E., 1500 Gregg, Big Spring.
Porter, Melvin A., 811 Main St., Big Spring.
Sanders, J. Virgil, Box 191, Big Spring.
Sanders, Nell W., P. O. Box 191, Big Spring.
Swift, Edward V., 811 Main St., Big Spring.
Talbot, Milton W., Jr.,
811 Main St., Big Spring.
Thomas, Clyde E., Jr.,
Howard Co. Hosp. Fdn., Big Spring.
Williams, Frankie E.,
Big Spring State Hosp., Big Spring.
Williamson, Thos. J.,
411 E. 9th St., Big Spring.
Wilson, Iva G., 811 Main St., Big Spring.
Wood, G. Hardin, Permian Bldg., Big Spring.
Woodall, Jack M., P. O. Box 991, Big Spring.
Worthy, Louise Bennett,
Drawer 949, Big Spring.

NOLAN-FISHER-MITCHELL

Callan, Chester M., Box 488, Rotan.
Callan, Chester U., Rotan.
Chinn, John H., Jr.,
505 Chestnut, Colorado City.
Cowan, Seth B.,
505 Chestnut St., Colorado City.
Cowan, W. Kenneth,
505 Chestnut St., Colorado City.
Crymes, J. Melvin, Colorado City.
Fortner, Amos H., (Hon.), Sweetwater.
Gollihar, William P., Young Clinic, Sweetwater.
Hood, Francis T. N., Jr.,
301 Locust, Sweetwater.
Johnson, Bruce H., Loraine.
Johnson, Clark A.,
Young Medical Center, Sweetwater.
Johnson, J. Frank, Rotan.
Kantor, John R., Hailey at 13th, Sweetwater.
LeBleu, Bennie, P. O. Box 473, Rotan.
Loeb, Sam A., Sweetwater.
Logsdon, Harry A., (Hon.),
Box 1068, Colorado City.
Peters, Roland O., Sweetwater.
Price, Robert L., Sweetwater.
Rhode, Oscar E., 505 Chestnut, Colorado City.
Rhode, William S.,
505 Chestnut St., Colorado City.
Richardson, James K., 1301 Hailey, Sweetwater.
Rudd, Laurence H., Box 1266, Colorado City.
Supowit, S. F., 500 Oak St., Sweetwater.
Terry, Joseph C., Loraine.
Wilkinson, Robert T., Roby.
Young, James W., (Hon.),
1401 Hailey, Sweetwater.
Young, Tom D., Sweetwater.

THIRD DISTRICT

Dr. William H. Gordon, Lubbock, Councilor

ARMSTRONG-DONLEY-CHILDRESS-COLLINGSWORTH-HALL-WHEELER

Blackketter, Donald E.,
113 East 2nd St., Shamrock.
Butler, Robert G., Jr.,
Second & Ave. E, Childress.
Cariker, Fred H., Childress.
Carroll, William A., (Hon.), (dead),
Amarillo.
Chaffin, Curtis R., 206 N. Main, Shamrock.
Clark, Robert E., Jr., Box 111, Memphis.
Fox, Grover C., (Hon.), Childress.
Fox, Jack F., 801 Commerce St., Childress.
Gooch, James W., Shamrock.
Goodall, O. R., Memphis.
Holcomb, Carter, Wellington.
Jenkins, O. L., (Hon.),
9330 Forest Hills, Dallas.
Jernigan, James H., (Hon.), Childress.
Jeter, Perry R., (Hon.), Childress.
Jones, Charles B., Wellington.
Lester, Joseph K.,
2410 S. 35th St., Lincoln, Nebr.
Nicholson, Harold E., Sr., (Hon.), Wheeler.
Nicholson, Harold E., Jr., (dead), Wheeler.
Odom, James A., (Hon.), Box 706, Memphis.
Roberts, Oran M., 104 East Second, Shamrock.
Smith, George W., Adair Hospital, Clarendon.
Stevenson, Harold R., Memphis.
Townsend, Shell H., Childress.
Walker, Glenn R., Wheeler.
Watkins, Dale V., Wellington.
Westenburg, Jacobus J., Childress.
Zeigler, Ray L., 113 W. Second St., Shamrock.

DALLAM-HARTLEY-SHERMAN-MOORE

Askins, J. Robert, Jr., 723 Bliss Ave., Dumas.
Coventry, William V., 115 W. 5th St., Dumas.
Cunningham, John H.,
201 Texas Blvd., Dalhart.
Elston, Fredrick A., (Int.),
7231 Edgemore Dr., Houston.
Goulding, Joseph C., 1215 N. Ange, El Paso.
Gutekunst, Roscoe A., 201 Texas Blvd., Dalhart.
Hull, Ivan W., 1324 Keeler, Dalhart.
Meredith, Duane W., 214 E. 5th St., Dumas.
Moore, Victor R., Box 1108, Dalhart.
Pieratt, Karl W., 124 Amherst, Dumas.
Purgason, John R., Box 535, Dumas.
Reed, Emil P., 101 Binkley St., Dumas.
Richardson, O. J., 201 Bliss, Dumas.
Smith, Paul E., Stratford.
Wright, Byron W., 214 E. 5th St., Dumas.

DEAF SMITH-PARMER-CASTRO-OLDHAM-SWISHER

Anderson, George H., Jr., Box 928, Friona.
Barnett, Lewis B., Box 986, Hereford.
Beene, Bruce G., 343 Miles Ave., Hereford.
Beyer, Lawrence A., Box 352, Hereford.
Bischoff, Harold W., P. O. Box 488, Dimmitt.
Burk, Houston M., 200 N. Austin, Tulia.
Childress, Wm. B., 118 S. W. 1st St., Tulia.
Cogswell, Ronald E., Dimmitt.
Cranfill, General Lee, Box 307, Friona.
Foreman, Lee S., Tulia.
Grubbs, Roy J., Box 352, Hereford.
Hicks, Clarence E., 343 N. Miles, Hereford.
Kempe, William W., 1511 N. Beckley, Dallas.
Mims, Arthur T., Hereford.
Nobles, Millard W.,
111 N. Knight St., Hereford.
Page, Carl F., Box 188, Dimmitt.
Richards, Fred V., Tulia.
Spence, Joseph W., Jr., Box 188, Dimmitt.
Spring, Floyd Lee, Friona.
Spring, Paul L., Friona.
Stewart, Evans P., Tulia.
Watley, Vernon T., Box 402, Vega.
Wills, Ralph R., Hereford.

HALE-FLOYD-BRISCOE

Benzon, George H., III,
801 West 8th St., Plainview.
Bublis, Mary E., 1001 W. Ninth, Plainview.
Bublis, Norbert J., 801 West 8th, Plainview.
Crum, Kenneth O., 1107 Xenia St., Plainview.
Davis, Jeff H., 220 St. Louis, Plainview.
Dils, Grover C., Caliente, Nevada.
Dye, Mary R., 312 Skaggs Bldg., Plainview.
Foster, Dee R., Box 245, Hale Center.
Freeman, Ray W., Hale Center.
Glenn, Thomas L.,
Lockney Gen. Hosp., Lockney.
Hansen, J. Harvey, (Hon.),
903 Milwaukee, Plainview.
Harvis, Herman J., 708 Denver, Plainview.
Heye, Randall G.,
801-813 W. 8th St., Plainview.
Hill, Clarence T., Jr.,
801 W. 8th St., Plainview.
Horn, Joe J., 220 St. Louis St., Plainview.
Howell, McKinley,
214 Skaggs Bldg., Plainview.
Jackson, Carl C., Box 1095, Plainview.
Johnson, Gilmer B., Jr.,
211 Skaggs Bldg., Plainview.
Long, Dorothy C., Skaggs Bldg., Plainview.
Long, John C., 801 W. 8th, Plainview.
Mangold, William J.,
Lockney Gen. Hosp., Lockney.
McCarthy, Eugene C., 220 St. Louis, Plainview.
Mitchell, Robert H.,
204 Skaggs Bldg., Plainview.
Nichols, E. O., Jr., 215 W. Alpine, Plainview.
Nichols, E. O., Sr., 1402 W. 11th, Plainview.
O'Grady, Joseph A.,
715 Houston St., Plainview.

Reed, William L., 801 W. Elm, Plainview.
Roberts, Roy R., 801 W. 8th St., Plainview.
Robins, James L., 801 West 8th St., Plainview.
Schlecte, Marvin C., 714 W. 7th, Plainview.
Smith, Landria C., 217 Skaggs Bldg., Plainview.
Snyderman, Henry,
Plainview Hospital, Plainview.
Thomas, W. Ralph, 220 St. Louis, Plainview.
Wagner, Gerald W.,
321 Skaggs Bldg., Plainview.
Williams, Pat, Lockney Gen. Hosp., Lockney.
Williams, Russell K.,
805 W. 8th St., Plainview.
Woods, H. S., Jr., 715 Houston St., Plainview.

HARDEMAN-COTTLE-FOARD-MOTLEY

Archer, James T., Box 1178, Spur.
Brooks, Walter A., 404 Mercer St., Quanah.
George, Joseph M., Quanah.
Harmon, Franklin C., Jr., Box 818, Paducah.
Howard, James C., Box 789, Chillicothe.
Hughes, John F., Spur.
Kralicke Martin M., 200 N. First, Crowell.
McDaniel, Robert R., Quanah.
Pate, Clarence C., Paducah.
Salkeld, Phil L., Quanah.
Sitta, Raymond E.,
607 E. Apache, Farmington, N. Mex.
Stanley, James S., Matador.
Stapp, Walter H., Box 146, Crowell.
Taylor, John M.,
3005 Ridgeway Dr., Metaire, La.
Todd, Eugene R., 404 Mercer St., Quanah.
Traweek, Albert C., Jr.,
P. O. Box 296, Matador.

LAMB-BAILEY-HOCKLEY-COCHRAN

Barnes, Elbert D., 1212 Houston St., Levelland.
Campbell, Dale P., 409 Austin, Levelland.
Davis, J. Walker, Cochran Co. Hosp., Morton.
Dupre, John D., 1212 Houston, Levelland.
Edgar, George V.,
1209-B Houston St., Levelland.
Fain, J. Randel, Box 112, Littlefield.
Faust, Fredric B., 3914 W. Lawn, Amarillo.
Green, Lathaggar, Jr., Box 368, Muleshoe.
Harrison, Robt. Joe, 409 Austin St., Levelland.
Hinckley, Herbert M.,
Med. Arts Hosp., Littlefield.
Janes, Fred W., Box 112, Littlefield.
Lee, Billie H., 200 E. Grant Ave., Morton.
Maurer, Ralph E., Box 347, Littlefield.
McSpadden, Wilton B.,
708 W. First St., Muleshoe.
Nowlin, William C., Box 112, Littlefield.
Phillips, Claurice M., 1212 Houston, Levelland.
Reid, Raymond A.,
Phillips-Dupre Hosp., Levelland.
Renegar, James G., 611 Ave. D, Levelland.
Santos, Ray E., West Plains Hosp., Muleshoe.
Shotwell, Ira T., Jr., Box 1031, Littlefield.
Sosa, Robert, 500 Littlefield Dr., Littlefield.
Stafford, Delmon J., Box 632, Littlefield.
Still, Oscar W., Box 112, Littlefield.
Walsh, Andrew L., 611 Avenue D, Levelland.

LUBBOCK-CROSBY

Adams, Erie Dell, 3801 19th St., Lubbock.
Anderson, William F., 1312 Main St., Lubbock.
Ansley, Leslie R., 3615 21st St., Lubbock.
Arnett, Sam C., Jr., 2609 19th St., Lubbock.
Arnold, Robert N., 1910 Knoxville, Lubbock.
Arrington, Joe O., Jr.,
2022 Broadway, Lubbock.
Barsh, Albert G., 2010 Broadway, Lubbock.
Batson, Carey B., 1315 Tenth St., Lubbock.
Baugh, William L., (Hon.),
1302 Main St., Lubbock.
Birdsong, William F., Muleshoe.
Blake, Emerson M., 1910 Knoxville, Lubbock.
Boling, John S., 1910 Knoxville, Lubbock.
Bronwell, Alvin W., 3801 19th St., Lubbock.
Canon, Robert T., 1312 Main St., Lubbock.
Carnrick, Millard, Jr.,
701 College Ave., Lubbock.
Carr, Robert L., 2602 Avenue Q, Lubbock.
Carter, John E., Box 1675, Post.
Cashion, William R., 2010 Broadway, Lubbock.
Chaffin, Walter T. C., 3615 19th St., Lubbock.
Chalk, John R., 802 Avenue N, Lubbock.
Chatman, Joseph A., 2303 Cedar, Lubbock.
Clark, Doyce M., 2615 19th St., Lubbock.
Clark, Vester V., (Hon.),
4716 22nd St., Lubbock.
Cobb, John L., 4412 W. 17th St., Lubbock.
Cole, Richard D., 1910 Knoxville, Lubbock.
Cone, Hubert E., 3302 46th St., Lubbock.
Croom, William S., 3801 19th St., Lubbock.
Curtis, Harrison E., 1910 Knoxville, Lubbock.
Daniel, Arthur L., 2613 34th St., Lubbock.
DeLaney, Robert M., 2609 19th St., Lubbock.
Donaldson, J. D., Jr., 3213 42nd St., Lubbock.
Douglas, Richard C., 1318 Main St., Lubbock.
Dunn, Jack, Jr., 1910 Knoxville, Lubbock.
Dunn, Sam G., 1312 Main St., Lubbock.
Duran C. Armando, Box 601, Lubbock.
Elkins, Clyde F., 1626 15th St., Lubbock.
Ellis, Noel A., 2602 Avenue Q, Lubbock.
English, Otis W., 1312 Main St., Lubbock.
Evans, William G., 1910 Knoxville, Lubbock.
Ewing, Mahon M., 2609 19th St., Lubbock.
Fiel, Charles A., 3701 19th St., Lubbock.
Forsythe, Ted H., 2303 56th St., Lubbock.
Garrett, Harold K., 1312 Main St., Lubbock.
Golightly, Chester G., 1312 Main St., Lubbock.
Goodwin, Frank C., 701 College Ave., Lubbock.
Gordon, William H., 2022 Broadway, Lubbock.
Hale, Lee E., 802 Avenue N, Lubbock.
Hall, James T., 511 College Ave., Lubbock.
Hancock, Howard R., 2615 19th St., Lubbock.
Hand, Orra Robert, 1910 Knoxville, Lubbock.
Haney, Edward L., (Inac.), Ralls.
Harris, Joseph R., Jr., 1211 Ave. O, Lubbock.
Healy, Maurice J., 1910 Knoxville, Lubbock.
Hendon, Robert G., Jr.,
1910 Knoxville, Lubbock.
Hess, Wallace I., 3201 42nd St., Lubbock.
Hewitt, Archie Lee, 1910 Knoxville, Lubbock.
Holmes, T. H., Jr., 3060 34th St., Lubbock.
Houser, O. Wayne, Box 356, Crosbyton.
Hudgins, Frank W., 3801 19th St., Lubbock.
Hull, O. Brandon, 2022 Broadway, Lubbock.
Hunt, Ewell L., 1312 Main St., Lubbock.
Hutchinson, Ben B., 3801 19th St., Lubbock.
Isham, A. Chapman, 2012 Nashville, Lubbock.
Jaynes, Stan H., 825 W. Lubbock St., Slaton.
Jenkins, B. Arthur, 1910 Knoxville, Lubbock.
Johnson, Lowell S., 1910 Knoxville, Lubbock.
Jones, Elmer W., 3415 20th St., Lubbock.
Jones, Glenn M., 3615 19th St., Lubbock.
Kallina, Frederick P., Tech. Infirmary, Lubbock.
Keffler, Richard A., 3615 19th St., Lubbock.
Key, Olan, 2609 19th St., Lubbock.
Krueger, Julius T., 1910 Knoxville, Lubbock.
Krueger, Julius T., Jr.,
1910 Knoxville, Lubbock.
Lane, Ralph, 1312 Main St., Lubbock.
Lewis, Richard Q., 3611 21st St., Lubbock.
Lewis, Royce C., Jr.,
1910 Knoxville St., Lubbock.
Locke, John C., P. O. Box 456, Slaton.
Long, William H., 3615 19th St., Lubbock.
Loveless, J. Elbert, 3801 19th St., Lubbock.
Loveless, Roy G., 2609 19th St., Lubbock.
Lovings, Franklin L., 2609 Hickory, Lubbock.
Lunceford, Tennie Mae,
1910 Knoxville, Lubbock.
Malone, Frank B., 2609 19th St., Lubbock.
Mantooth, Walter B., Jr.,
3801 19th St., Lubbock.
Marable, Gerald L., 3414B Avenue H, Lubbock.
Matthews, James R., Box 1437, Post.
Mattison, Myron D., 3621 21st St., Lubbock.
Mayfield, Ivan G., 1711 30th St., Lubbock.
McCarty, Robert H.,
1910 Knoxville Ave., Lubbock.
McClure, Edwin E., 701 College Ave., Lubbock.
McDaniel, Bennie O., Muleshoe.
Miller, John T., Jr.,
Great Plains Bldg., Lubbock.
Miller, Pauline A., 2609 19th St., Lubbock.
Monk, Norman A.,
511 College Drive, Lubbock.
Monte, Melville R., 3408 54th St., Lubbock.
Montgomery, Clifford L.,
1318 Main St., Lubbock.
Moore, Robert, 3801 19th St., Lubbock.
Morris, James G., 1312 Main St., Lubbock.
Moss, Charles B., 1318 Main St., Lubbock.
Moss, Ennis E., Methodist Med. Bldg., Lubbock.
Nalle, Ernest, Jr., 1312 Main St., Lubbock.
Nash, Charles H., Jr.,
1910 Knoxville, Lubbock.
Nemir, Stuart S., Jr., 3615 21st St., Lubbock.
O'Loughlin, Richard K.,
3801 19th St., Lubbock.
Pappas, Patrick H., 3621 21st St., Lubbock.
Payne, Clifford E., 2415 6th St., Lubbock.
Payne, Glen B., 250 W. Lubbock, Slaton.
Pennington, Hugh A., 2613 34th St., Lubbock.
Pigford, Charles A., 1202 Jarvis St., Lubbock.
Poole, Warren B., 3060 34th St., Lubbock.
Porres, Felipe D., 4906 17th St., Lubbock.
Porres, Norma E., Box 998, Lubbock.
Ratcliff, Charles E., 3801 19th St., Lubbock.
Rhoades, Dale R., Crosbyton.
Riddel, Roy L., Jr., 1318 Main St., Lubbock.
Rountree, John B., Jr., 2615 19th St., Lubbock.
Rutledge, Randolph, 3801 19th St., Lubbock.
Ryburn, Frank M., Jr., 3621 21st St., Lubbock.
Schmidt, Elwood L., 123 W. Lubbock, Slaton.
Selby, John H., 1910 Knoxville, Lubbock.
Seymour, Fred W., 1302 Main St., Lubbock.
Shannon, Emmet, 3801 19th St., Lubbock.
Sheffield, Roy S., 3707 48th St., Lubbock.
Shepard, Herbert L., Med. Arts Clinic, Lubbock.
Slemmons, Theodore M., Muleshoe.
Smith, Jerome A., Great Plains Bldg., Lubbock.
Smith, Lynwood B., 1910 Knoxville, Lubbock.
Smith, Richard D., 1318 Main St., Lubbock.
Smith, Roy L., 1910 Knoxville, Lubbock.
Smith, William C., 3801 19th St., Lubbock.
Snodgrass, Milo R., Crosbyton.
Snow, Wister C., (Inac.), Ralls.
Spikes, Lowell W., Ralls.
Stewart, Allen T., 1318 Main St., Lubbock.
Stiles, James Hooper, 3801 19th St., Lubbock.
Stirman, Jerry A., 1910 Knoxville, Lubbock.
Storrs, Loyd A., 3801 19th St., Lubbock.
Stout, Ruth Louise, 3702 21st St., Lubbock.
Surman, Arnold C., (Hon.), Post.
Talbert, Thomas L., 123 W. Lubbock, Slaton.
Taylor, Otis, Jr., 511 College, Lubbock.
Tubbs, Harry A., Post.
Ullom, William S., 4122 34th St., Lubbock.
Upshaw, Leon R., 1312 Main St., Lubbock.
Wallace, Grady M., 3801 19th St., Lubbock.
Warshaw, Harold, 3801 19th St., Lubbock.
Watkins, Mina D., 1312 Main St., Lubbock.
Williams, David C., (Hon.), Post.
Yarbrough, Ocie Carl,
5009 College Ave., Lubbock.

POTTER-RANDALL

Archer, John R., 2603 Henning, Amarillo.
Aronson, Sam J. R., (dead), Amarillo.
Askew, Wesley L., Jr., 1422 Tyler, Amarillo.
Blackwell, Ben T., 705 Fisk Bldg., Amarillo.
Bordelon, Howard M.,
2414 Line Ave., Amarillo.
Boswell, Leta N., Canyon.
Bryan, E. Kay, 819 Martin Road, Amarillo.
Bryan, J. Manly, 608 16th St., Canyon.
Budd, Robert G., P. O. Box 7087, Amarillo.
Budd, Wilbur Q., 802 Rusk, Amarillo.
Butler, William H., Jr.,
1601 N.W. 20th, Amarillo.
Campbell, William J.,
2209 W. 7th Ave., Amarillo.
Carroll, J. Ralph, 2209 W. 7th Ave., Amarillo.
Chase, Gaylord R., 1422 Tyler St., Amarillo.
Chastain, Richard L.,
2209 W. 7th Ave., Amarillo.
Churchill, T. Preston, Box 710, Amarillo.
Citron, Ralph, 2209 W. 7th, Amarillo.
Cole, Marion W., V. A. Hosp., Amarillo.
Cooper, Orris V.,
1409 N. Bonham St., Amarillo.
Crume, John J., (Hon.),
Herring Hotel, Amarillo.
Crumley, Fred J., 2428 W. 8th, Amarillo.
Daugherty, Roy, 4002 S. Washington, Amarillo.
Davis, Frances E., 2301 West 7th, Amarillo.
Davis, Margaret M., 1422 Tyler St., Amarillo.
Denko, John V., Box 1110, Amarillo.
Devanney, Louis R.,
2209 W. 7th Ave., Amarillo.
Dine, William C., 1422 Tyler St., Amarillo.
Dravin, Isadore, 2209 W. 7th Ave., Amarillo.
Duke, Tom W., 1422 Tyler St., Amarillo.
Duncan, Frank B.,
339 Amarillo Bldg., Amarillo.
Duncan, Robert A.,
339 Amarillo Bldg., Amarillo.
Ellis, J. Victor, 2301 W. Seventh, Amarillo.
Erdmann, Ralph R., Box 3274, Amarillo.
Flamm, Kenneth R.,
2209 W. 7th Ave., Amarillo.
Gallagher, Robert P., (Hon.), (dead),
Amarillo.
Garre, Peter R., 1422 Tyler St., Amarillo.
Gilkerson, Nan L., (Inac.),
Fisk Bldg., Amarillo.
Glenn, James C., 1501 W. 10th St., Amarillo.
Glenn, William A., 2412 Line Ave., Amarillo.
Goldston, Alton B.,
1501 W. 10th St., Amarillo.
Greer, Rex E., 412 Barfield Bldg., Amarillo.
Haralson, Prescott H.,
2750 Duniven Cir., Amarillo.
Harkleroad, Frank S., 616 N. Polk, Amarillo.
Hatchett, Capres S., 800 Rusk St., Amarillo.
Hegedus, William J.,
2714 W. 10th St., Amarillo.
Hyde, Robert F., 2714 W. 10th St., Amarillo.
Intress, Robert H., Box 3572, Amarillo.
Jackson, Harvey K., Box 3275, Amarillo.
Jacobson, Merlin E.,
V. A. Hosp., Wichita, Kansas.
Jarrett, Robert P., Canyon.
Johnson, H. Fred, 2308 W. 8th St., Amarillo.
Johnson, James L.,
2209 W. 7th Ave., Amarillo.
Johnson, Jere B., 1501 W. 10th St., Amarillo.
Johnson, L. G., 5506 Tawney St., Amarillo.
Jones, Billy Rual, 708 Monroe St., Amarillo.
Jones, Elmer K., 604 W. 8th Ave., Amarillo.
Jordaan, John D., (Inac.),
Box 1411, Amarillo.
Kelly, Francis J., 810 Bryan St., Amarillo.
Klingensmith, Wm., Jr., 1422 Tyler, Amarillo.

Klingensmith, Wm. R., (Inac.),
1413 Lipscomb, Amarillo.
Lara, Jorge Y., 2301 W. 7th, Amarillo.
Latson, Harvey H.,
2209 W. 7th Ave., Amarillo.
Laur, William E., 608 W. 8th, Amarillo.
Lemmon, J. Ralph,
2714 West 10th Ave., Amarillo.
Lipscomb, Joe L., 2414 W. 9th, Amarillo.
Lokey, Early B., 719 Austin, Amarillo.
Loving, Dan H., 611 Fisk Bldg., Amarillo.
Marsalis, Don S., 1422 Tyler, Amarillo.
Masters, Bert A.,
2724 Westhaven Vil., Amarillo.
McCaleb, Morgan H., 1422 Tyler, Amarillo.
McClelland, Robert N.,
Neblett Hosp. & Cl., Canyon.
McKay, Edward D.,
2300 W. 7th Ave., Amarillo.
Mears, Edward J., 1501 W. 10th St., Amarillo.
Mok, Wa T., 2300 W. 7th, Amarillo.
Moore, G. Dudley, Jr., Box 35, Canyon.
Mullins, William B.,
2308 W. 8th St., Amarillo.
Murphy, Weldon O.,
2300 W. 7th Ave., Amarillo.
Naslund, Edward G., 1009 W. 10th, Amarillo.
Neblett, Robert A.,
Neblett Hosp. & Cl., Canyon.
Nester, Chas. R., Canyon.
Oles, C. Patrick, 1422 Tyler, Amarillo.
Owens, Guy, 2209 W. 7th Ave., Amarillo.
Parker, William L.,
133 N. Rosemont, Amarillo.
Patton, David M., 703 Harrison St., Amarillo.
Patton, Louis K., 703 Harrison St., Amarillo.
Patton, Walter S., 703 Harrison St., Amarillo.
Payne, Ralph B., 608 W. 8th St., Amarillo.
Pennal, Hugh A., 2209 W. 7th St., Amarillo.
Pickett, John M.,
2714 West 10th Ave., Amarillo.
Potter, Wilkes A., 2007 West 7th, Amarillo.
Powers, Evelyn G., Fisk Bldg., Amarillo.
Powers, George L., Fisk Bldg., Amarillo.
Price, William T., Jr.,
2209 W. 7th Ave., Amarillo.
Pronko, Michael J., 2300 W. 7th St., Amarillo.
Puckett, Howard E.,
2209 W. 7th Ave., Amarillo.
Quintanilla, Jaime, 807B Lamar, Amarillo.
Randall, Albert G., Box 1971, Amarillo.
Reed, Holley W., 2414 W. 9th, Amarillo.
Robberson, Jason H., 1422 S. Tyler, Amarillo.
Robberson, Joe Frank, (Int.),
9941 Bassoon, Houston.
Rowley, Elmer A.,
2209 W. 7th Ave., Amarillo.
Royse, George T., 603 Fisk Bldg., Amarillo.
Russell, Woolworth, 2710 W. 10th, Amarillo.
Sadler, Charles B., 915 W. 8th Ave., Amarillo.
Scott, Wilbert E., 706 Monroe St., Amarillo.
Smith, G. Ernestine, 814 Lamar St., Amarillo.
Stout, William W.,
2209 W. 7th Ave., Amarillo.
Streit, August J., 2300 W. 7th Ave., Amarillo.
Swindell, Raymon R.,
2400 Line Ave., Amarillo.
Thomas, Edward F., 600 W. 8th, Amarillo.
Vaughan, John H., 322 Sunset, Amarillo.
Vineyard, Roy L., 1801 Harrison, Amarillo.
Waddill, George M., Jr.,
2308 W. 8th, Amarillo.
Walkes, Ernest E.,
8805 W. 4th Ave., Lakewood, Colo.
Watkins, Walter C., 606 W. 8th, Amarillo.
Werner, Jan R., 2307 W. 7th, Amarillo.
Wertz, Royal F., 800 Rusk St., Amarillo.
Wheir, William H., 2400 Line Ave., Amarillo.
White, Jesse B., 2209 W. 7th Ave., Amarillo.
Winsett, Amos E., 611 Fisk Bldg., Amarillo.
Winsett, E. Merrill, 611 Fisk Bldg., Amarillo.
Witcher, Jones E., 2300 Line Ave., Amarillo.
Wolf, Horace L., P. O. Box 2068, Amarillo.
Wolfson, Charles, 2718 W. 10th, Amarillo.
Wright, Norman E., 1422 Tyler, Amarillo.
Wyatt, George W., 2109 W. 7th St., Amarillo.
Yeakel, S. Victor, 2400 Line Ave., Amarillo.
Zientek, Ralph J., 3106 Harmony, Amarillo.

TOP O' TEXAS
(GRAY-HANSFORD-HEMPHILL-LIPSCOMB-ROBERTS-OCHILTREE-HUTCHINSON-CARSON)

Ashby, Charles H., 1701 N. Hobart St., Pampa.
Barksdale, William C.,
510 N. Weatherly, Borger.
Bellamy, Russell M., 1002 N. Hobart, Pampa.
Black, Crawford H., 505 N. Weatherly, Borger.
Brown, R. Malcolm, Florida & Hobart, Pampa.
Donaldson, Joe R., Hughes Bldg., Pampa.
Elder, John F., 600 W. Kentucky, Pampa.
Ellis, Neely Joe, Combs Worley Bldg., Pampa.
Fabian, Harold F., Box Q, McLean.
Falkenstein, Richard D.,
600 W. Kentucky, Pampa.
Friedman, Hyman, 1008 Megert Center, Borger.
Gates, Joseph W., P. O. Box 1421, Pampa.
Gates, Philip A., Box 1421, Pampa.
Gray, Elkanah B., 108 E. Broadway, Stinnett.
Hampton, Dan E., 207 S. McGee, Borger.
Hampton, Raymond M., M & S Clinic, Pampa.
Hamra, Henry M., 100 S. McGee, Borger.
Harvey, T. DeWitt, 808 W. Francis, Pampa.
Hays, Harvey, Jr., 412 S. Main St., Borger.
Heerwagen, Paul K., Jr.,
Phillips Med. Cen., Phillips.
Hollis, Lynn E., 1 S. Addinsell, Phillips.
Holmes, Robert L., Jr., 315 Baylor, Borger.
Hrdlicka, George R., 300 Hughes Bldg., Pampa.
Huff, Oscar, (Hon.), 1116 Christine, Pampa.
Ingham, Mahlon E., 500 W. 3rd, Borger.
Johnson, J. Bluford,
Sanford Clinic Hosp., Perryton.
Jones, W. Calvin, 312 Rose Bldg., Pampa.
Kelley, Frank W., 516 W. Kentucky, Pampa.
Kengle, George L., Perryton.
Key, Julian M., Box 1782, Pampa.
Kleeberger, Roland L., Box 545, Spearman.
Knowles, Joe H., 1063 Coronado Cir., Borger.
Lang, Carl M., Hughes Bldg., Pampa.
Laycock, Raymond W.,
516 W. Kentucky, Pampa.
Massad, Woodrow W., 100 S. McGee, Borger.
McDaniel, MacField, Box 1782, Pampa.
Monroe, Carroll D., 514 S. Main, Perryton.
Moore, Robert W., 1832 Fir St., Pampa.
Morris, Ernest H., Canadian.
Overton, Marvin C., Jr., Hughes Bldg., Pampa.
Powell, Paul H., Jr., 500 W. Third St., Borger.
Prewit, Rex D., 500 W. Third St., Borger.
Sanford, Roy K., Perryton.
Snyder, Edward H., (Hon.), Canadian.
Snyder, Rush A., 405 Main St., Canadian.
Stephens, Milton M., 412 S. Main, Borger.
Stephens, Walton G., 412 S. Main, Borger.
Stewart, Allen T., Jr., Box 325, Spearman.
Vendrell, Felix J., Pampa Clinic, Pampa.
Voet, Henriette H.,
114 W. Grand Ave., Borger.
Wheeler, Jim E., 1063 Coronado Cir., Borger.
Williams, Edward S.,
300 Hughes Bldg., Pampa.

FOURTH DISTRICT

Dr. Oren H. Chandler, Ballinger, Councilor

BROWN-COMANCHE-MILLS-SAN SABA

Bowden, A. M., (Hon.), May.
Bullard, Chester C., (Hon.),
1616 Austin Ave., Brownwood.
Cadenhead, Ernest F.,
2401 Coggin Ave., Brownwood.
Childress, Marvin A., Goldthwaite.
Coleman, Rogers K.,
308 W. Chandler, Brownwood.
Cruzcosa, Catalino G.,
Childress Cl. Hosp., Goldthwaite.
Cutbirth, Seale T.,
308 W. Chandler, Brownwood.
Everett, Jerry J.,
2400 Coggin Ave., Brownwood.
Farley, Frederick W., San Saba.
Felts, Richard C., San Saba.
Gold, Philip S., Brownwood.
Gray, Charles W., (Hon.), Comanche.
Hallum, Roy G.,
409 E. Adams St., Brownwood.
Lobstein, Henry L.,
1319 Austin Ave., Brownwood.
Locker, S. Braswell, 1501 11th St., Brownwood.
Mayo, Oscar N., Box 790, Brownwood.
McFarlane, Joe R.,
Citizens Bank Bldg., Brownwood.
McGowan, Patrick T.,
Med. Arts Clinic, Brownwood.
Ory, Lee K., Comanche.
Snyder, Ned, Jr., Coggin at 12th, Brownwood.
Spencer, Fred D., Jr.,
308 West Chandler, Brownwood.
Stephens, Joe B., P. O. Drawer 38, Bangs.
Sweet, William O.,
Citizens Natl. Bank Bldg., Brownwood.
Walker, James B. N., Brownwood.
Wheelis, Paul M.,
Medical Arts Bldg., Brownwood.

COLEMAN

Aston, S. N., (Hon.), Coleman.
Burke, Francis M., 309 S. Pecos, Coleman.
Duke, Jean D., 208 W. College, Coleman.
Kemper, R. F.,
Coleman Mutual Bldg., Coleman.
Lovelady, Roy R., 309 S. Pecos St., Coleman.
Mann, Morris D., 301 W. Pecan, Coleman.
Moody, Charles O., 301 W. Pecan St., Coleman.
Rehngren, J. Philip, 1400 Fae St., Santa Anna.
Weaver, Manly E.,
Coleman Co. Bank Bldg., Coleman.
Young, Josephus C., 205 W. Elm, Coleman.

CRANE-UPTON-REAGAN

Cooper, James L., McCamey.
Gossett, James D., Rankin.
Holt, William D., Box 1325, McCamey.
Maynard, Billy J., Box 1145, Crane.
Nibling, Boyd, Box 1178, McCamey.
Nunnally, Cleon S., 703 Depot, Big Lake.
Terry, John E., Box 1145, Crane.
Wright, John L., Jr., Box 847, Big Lake.

KIMBLE-MASON-MENARD-McCULLOCH

Anderson, James P., 1304 S. High St., Brady.
Anderson, James S., (Hon.), Brady.
Bodenhamer, James G., Mason.
Cole, James R., Box 426, Mason.
Hallum, B. A., Jr., Brady.
Hanus, Joseph J., Austin State Hosp., Austin.
Hartgraves, Hallie, (Hon.),
Kemper Hall, Kenosha, Wisc.
Hays, Aaron R., (Hon.), Brady.
Hays, Robert D., Brady.
Hinchman, A. Warren, Brady.
Jordan, Dowdell W., Brady.
Land, Wm. M., (Hon.), Lohn.
McCulloh, Albert M., Brady.
Ricks, Glenn H., Brady.
Westphal, Herbert M., Menard.

RUNNELS

Bailey, Charles F., Ballinger.
Chandler, Oren H., Ballinger.
Cook, James T., 804½ Hutchins, Ballinger.
Cottle, Kenneth E., 804½ Hutchins, Ballinger.
Dixon, James W., (Hon.), Box 30, Winters.
Green, John E., Jr., Ballinger.
Griffin, John E., Box 898, Winters.
Jennings, Thomas V., (Hon.), Winters.
McCreight, Henry H., Box R, Winters.
Rives, C. T., Winters.
Shiller, John J., Rowena.

TOM GREEN EIGHT
(TOM GREEN-COKE-CROCKETT-CONCHO-IRION-STERLING-SUTTON-SCHLEICHER)

Adams, M. Dean, (Mil.),
2850 Madison, Yuma, Arizona.
Alexander, Eugene P.,
2021 W. Beauregard, San Angelo.
Allison, Richard F.,
McKnight State San., Sanatorium.
Anderson, Hiram M.,
224 E. Harris, San Angelo.
Anderson, Wilson D.,
123 W. Harris, San Angelo.
Arledge, Robert M.,
203 E. Harris, San Angelo.
Ballard, John E.,
Shannon Mem. Hosp., San Angelo.
Barry, Douglas J.,
115 E. Beauregard, San Angelo.
Bolen, John G., Med. Arts Bldg., San Angelo.
Boster, Raymond G.,
510 W. Beauregard, San Angelo.
Brask, H. Kermit, 1411 Shafter, San Angelo.
Brauns, Wilhelm H.,
234 W. Beauregard, San Angelo.
Browne, Charles F., Sonora.
Bunyard, Joseph A.,
S. A. Natl. Bank Bldg., San Angelo.
Burk, Houston W., Box L, Eden.
Butner, Wendell B.,
Med. Arts Bldg., San Angelo.
Byars, Perry J. C., Jr.,
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234 W. Beauregard, San Angelo.
Coleman, T. Gabe,
510 W. Beauregard, San Angelo.
Cornelison, Joe L.,
510 W. Beauregard, San Angelo.
Diserens, H. William,
234 W. Beauregard, San Angelo.
Dodson, Dwain F.,
111 E. Harris Ave., San Angelo.
Eckhardt, Gus F.,
510 W. Beauregard, San Angelo.
Engelking, Chas. F.,
234 W. Beauregard, San Angelo.
Everhart, Merrill W.,
111 E. Harris Ave., San Angelo.
Finks, Robert M., Clinic Hospital, San Angelo.
Franken, Robert,
408 Chadbourne Bldg., San Angelo.
French, Cecil M., 339 W. Harris, San Angelo.
Gainer, Marthalyn J.,
612 E. Parkway, San Angelo.
Gainer, Sam H., Med. Arts Bldg., San Angelo.
Gaspar, Geza,
2650 University Ave., San Angelo.

Harris, John R., Bronte.
Harris, Orion W.,
Box 98, State Sanatorium, Ark.
Hayter, Dale W.,
234 W. Beauregard, San Angelo.
Hershberger, Lloyd R.,
Med. Arts Bldg., San Angelo.
Hollister, W. L., (Inac.),
202 E. Ave. J, San Angelo.
Hooker, James W.,
510 W. Beauregard, San Angelo.
Howell, John F., Sonora.
Hunter, Darlene L.,
2625 University, San Angelo.
Hunter, Thos. R., Jr.,
234 W. Beauregard, San Angelo.
Hutchins, F. Leon,
Med. Arts Bldg., San Angelo.
Irvine, Geo. N., Jr.,
510 W. Beauregard, San Angelo.
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Jones, Robert R.,
2015 W. Beauregard, San Angelo.
Kilman, William J.,
McKnight San., Sanatorium.
Knight, Maynard D.,
234 W. Beauregard, San Angelo.
Kunath, Carl A., 111 E. Harris, San Angelo.
Landrum, Charles K.,
111 E. Harris Ave., San Angelo.
Landy, Aaron E., 14 S. Jefferson, San Angelo.
Lewis, Aubrey L.,
123 E. Beauregard, San Angelo.
Martin, Scott H., 115 S. Park, San Angelo.
McCaw, Blanche M., 444 Preusser, San Angelo.
McCaw, William H.,
111 E. Harris, San Angelo.
McDaniel, John D., Robert Lee.
McIntire, Floyd T., 111 E. Harris, San Angelo.
McKnight, Joseph B., (Hon.),
2206 Dallas St., San Angelo.
Mee, Edmond L., 224 E. Harris, San Angelo.
Moon, Roy E.,
234 W. Beauregard, San Angelo.
Morse, Robert A.,
234 W. Beauregard, San Angelo.
Mowrey, Jack I., Box 548, Eldorado.
Nesrsta, George L., 111 E. Harris, San Angelo.
Nibling, George W., (Hon.),
1416 W. Harris, San Angelo.
Nylander, Calvin P.,
2102 Colorado, Apt. B, San Angelo.
Pilmer, Gordon A.,
Med. Arts Bldg., San Angelo.
Porter, William L.,
111 E. Harris, San Angelo.
Powers, Rufus L., 203 E. Harris, San Angelo.
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Read, John L., 111 E. Harris, San Angelo.
Reilly, Daniel R., 206 S. Bishop, San Angelo.
Ricci, Henry N., 602 S. Abe, San Angelo.
Round, Harry F., 201 E. Harris, San Angelo.
Round, Kye B., 201 E. Harris, San Angelo.
Rush, Clyde E., McKnight San., Sanatorium.
Schulkey, William E., (Hon.),
2460 Nasworthy Dr., San Angelo.
Schulze, Victor E.,
219 S. Magdalen, San Angelo.
Sessums, J. Valton,
635 S. Bishop St., San Angelo.
Sherrill, Lloyd H., Ozona.
Simon, Ralph E., Jr., Box 818, Ozona.
Smith, W. Lacey, 111 E. Harris, San Angelo.
Spencer, Francis M.,
111 E. Harris Ave., San Angelo.
Swann, William J., Sterling City.
Tandy, Hugh B., Ozona.
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206 E. Harris, San Angelo.
Williams, Harvey M.,
Med. Arts Bldg., San Angelo.
Wimpee, James D.,
510 W. Beauregard, San Angelo.
Windham, Robert E., (Hon.),
Christoval Road, San Angelo.
Winkelmann, Eugene C.,
2102 Pecos, San Angelo.
Womack, Clifford T., (Hon.),
Med. Arts Bldg., San Angelo.
Womack, James C.,
Med. Arts Bldg., San Angelo.
Womack, William T.,
Med. Arts Bldg., San Angelo.
Wood, Marion L., 224 E. Harris, San Angelo.

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1510 Pleasanton Rd., San Antonio.
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318 N. Santa Rosa, San Antonio.
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Albert, Monroe, 2714 S. Presa St., San Antonio.
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523 Greenwich Blvd., San Antonio.
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1018 S. Texas Bldg., San Antonio.
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205 Camden St., San Antonio.
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909 Nix Prof. Bldg., San Antonio.
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Bates, Leroy E., Jr., (Int.),
Lahey Clinic, Boston, Mass.
Beach, Asa, 205 Camden St., San Antonio.
Beck, Emma, (Inac.), Fredericksburg.
Bedolla, Miguel A., 706 S. Laredo, San Antonio.
Benavides, Jose M.,
424 International Bldg., San Antonio.
Bennett, Bruce H., (Mil.),
3510th USAF Hosp., Randolph AFB.
Bennett, Eaton W.,
P. O. Box 1840, San Antonio.
Benson, Otis O., Jr., (Mil.),
Sch. of Avn. Med., Brooks AFB.
Berchelmann, Adolph, (Inac.),
809 S. Laredo, San Antonio.
Berchelmann, August G.,
200 Donaldson, San Antonio.
Berchelmann, David A.,
809 S. Laredo St., San Antonio.
Berler, James M.,
601 Med. Prof. Bldg., San Antonio.
Bernard, George E.,
2911 S. New Braunfels, San Antonio.
Berry, Charles A., (Mil.),
Off. Surg. Gen. USAF, Washington 25, D. C.
Berry, Joe H., 516 Lexington, San Antonio.
Berry, Lloyd E., Jr.,
1100 Austin Hwy., San Antonio.
Biggar, James H., (Hon.),
242 Rockwood St., San Antonio.
Bishop, Elmer W.,
1911 Pleasanton Rd., San Antonio.
Blair, James R., Jr.,
516 Lexington Ave., San Antonio.
Block, William J.,
1111 Nix Prof. Bldg., San Antonio.
Bloom, Bernard H.,
308 Med. Prof. Bldg., San Antonio.
Blount, Robert E., (Mil.),
Brooke Army Hosp., Ft. Sam Houston.
Blumer, Max A., (Inac).,
3886 Beechwood Dr., Pittsburgh, Pa.
Boehs, Charles J.,
1119 Med. Arts Bldg., San Antonio.
Bohmfalk, John H.,
227 E. Hildebrand, San Antonio.
Boldt, John W., 3607 Wyoming, Houston.
Bondurant, William W., Jr.,
811 Nix Prof. Bldg., San Antonio.
Bonnet, Edith M., (Inac.),
616 Ogden Lane, San Antonio.
Borders, James L., (Mil.),
1605th USAF Hosp., New York, N. Y.
Borsheim, Raymond S.,
2608 N. Main Ave., San Antonio.
Bos, Norman C., (Mil.),
926 Fairchild, Lackland AFB.
Bosshardt, Carl E.,
1226 Nix Prof. Bldg., San Antonio.
Botkin, Rolan R., 3721 S. Presa, San Antonio.
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Bowen, Robert E., 111 Latch Dr., San Antonio.
Boyd, Everett M., (Int.),
Robt. B. Green Hosp., San Antonio.
Boysen, Arthur E.,
1520 McCullough, San Antonio.
Bradley, Roy W.,
120 Med. Prof. Bldg., San Antonio.
Brannon, Earl W., (Mil.),
Lackland AFB, San Antonio.
Brendel, William B.,
111 Gen. Krueger, San Antonio.
Breuer, Alfred, 525 Richmond, San Antonio.
Brewer, Dorothy, 124 Dallas St., San Antonio.
Britton, Howard A.,
402 Fulton Ave., San Antonio.
Brooks, Robert H., (Mil.),
Kelly AFB, San Antonio.
Brown, Alexander A., (Hon.),
233 Linda Dr., San Antonio.
Brown, Charles T.,
215 Blue Bonnet Blvd., San Antonio.
Brunner, G. Harmon,
306 Med. Prof. Bldg., San Antonio.
Brunner, Robbie Neeley,
306 Med. Prof. Bldg., San Antonio.
Burg, Edward M.,
805 Maverick Bldg., San Antonio.
Burk, Joseph E., 3203 San Pedro, San Antonio.
Burk, William E.,
1030 Med. Arts Bldg., San Antonio.
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1717 Nix Prof. Bldg., San Antonio.
Burton, Kenneth G.,
3506 S. New Braunfels, San Antonio.
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1031 Nix Prof. Bldg., San Antonio.
Buttery, James M.,
609 Med. Arts Bldg., San Antonio.
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110 E. Hermosa Dr., San Antonio.
Calvert, Hulon E.,
506 Med. Arts Bldg., San Antonio.
Campbell, Billy Gay,
3721 S. Presa St., San Antonio.
Campbell, Daniel C., Jr., (Mil.),
USAF Hospital, Lackland AFB.
Canales, Gregorio M.,
917 Nix Prof. Bldg., San Antonio.
Carter, James W., Jr.,
818 Nix Prof. Bldg., San Antonio.
Case, John B.,
625 Med. Arts Bldg., San Antonio.
Castle, Margaret R.,
545 New Moore Bldg., San Antonio.
Cayo, Edward A.,
923 Med. Arts Bldg., San Antonio.
Cayo, Ernest P.,(Inac.),
755 E. Mulberry, San Antonio.
Celaya, Albert, 124 Dallas St., San Antonio.
Celaya, Henry,
1211 Med. Arts Bldg., San Antonio.
Center, William M., 605 Belknap, San Antonio.
Champion, Albert N.,
1223 Med. Arts Bldg., San Antonio.
Chandler, Jack R.,
915 Med. Arts Bldg., San Antonio.
Chankin, Edgar D.,
525 Richmond, San Antonio.
Childers, Herschel N.,
7407 Broadway Ave., San Antonio.
Childers, Marvin A.,
7407 Broadway, San Antonio.
Christian, Thomas E.,
810 Med. Arts Bldg., San Antonio.
Chuang, Theodore H.,
Box 7206, Hackberry Sta., San Antonio.
Clark, A. Fletcher, Jr.,
506 Med. Prof. Bldg., San Antonio.
Clark, A. Fletcher, Sr.,
225 Med. Arts Bldg., San Antonio.
Clark, William B., (Mil.),
Sch. of Avn. Med., Brooks AFB.
Clifton, Collis B.,
303 Maverick Bldg., San Antonio.
Coates, Elmer T.,
401 W. Summit, San Antonio.

Cochran, J. Layton,
1220 Med. Arts Bldg., San Antonio.
Coffman, Graham M.,
625 Med. Arts Bldg., San Antonio.
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204 E. Rosewood Ave., San Antonio.
Conroy, Normabelle H.,
307 Wyndale, San Antonio.
Cook, Clara G.,
1008 Rigsby Ave., San Antonio.
Cook, Walter R.,
123 Ogden Lane, San Antonio.
Cooper, Elmer E., 505 Howard, San Antonio.
Cooper, Fred B., 2001 N. Main, San Antonio.
Cooper, Harold N.,
212 W. Laurel, San Antonio.
Cooper, Jean Head,
211 Richmond Ave., San Antonio.
Cooper, Melbourne J.,
211 Richmond Ave., San Antonio.
Copeland, Joseph B.,
1014 Med. Arts Bldg., San Antonio.
Cover, Ellen C., (Inac.),
Rt. 6, Box 297, San Antonio.
Cowles, Andrew G., (Hon.),
202 W. Kings Hwy., San Antonio.
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1030 Nix Prof. Bldg., San Antonio.
Creamer, Dick O.,
817 S. Texas Bldg., San Antonio.
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410 Med. Prof. Bldg., San Antonio.
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111 Pinecrest Blvd., San Antonio.
Culver, James F., (Mil.),
Sch. of Avn. Med., Brooks AFB.
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1108 Nix Prof. Bldg., San Antonio.
Day, Proctor W.,
Kelly AFB Disp., San Antonio.
Day, Richard T., (Mil.),
Sch. of Avn. Med., Randolph AFB.
DeCoursey, Elbert, Rt. 4, Box 86, San Antonio.
DeGasperi, Joseph A.,
146 Brandon Dr. W., San Antonio.
DeLeon, John J., 1418 Nix Bldg., San Antonio.
del Valle, Antonio F.,
P. O. Box 10067, San Antonio.
Dennett, M. Harley, Jr.,
730 Med. Arts Bldg., San Antonio.
Des Rochers, Jean B.,
Box 7206, Hackberry Sta., San Antonio.
Diseker, Thomas H.,
328 Med. Arts Bldg., San Antonio.
Dishon, Margaret P.,
2504 N. Main St., San Antonio.
Dishon, Neil H.,
2504 N. Main St., San Antonio.
Dittman, Charles H.,
411 Med. Arts Bldg., San Antonio.
Dodge, Donald T.,
1216 S. Texas Bldg., San Antonio.
Dominy, Dale E., (Mil.),
Lackland USAF Hosp., San Antonio.
Donaldson, James M., Jr.,
923 Nix Bldg., San Antonio.
Donop, Perry T.,
2020 Alamo Natl. Bank, San Antonio.
Dorbandt, Moss M.,
128 W. Commerce St., San Antonio.
Doyle, John L., 1525 Nix Bldg., San Antonio.
Dubrow, Donald N., (Mil.),
102 Samoth Dr., San Antonio.
Dufner, Romie M.,
3721 S. Presa St., San Antonio.
Dumas, Edward D.,
425 Med. Arts Bldg., San Antonio.
Duncan, Everett T.,
1009 S. Texas Bldg., San Antonio.
Dupre, Joseph R.,
2331 Vance Jackson, San Antonio.
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250 Gen. Hosp., Ft. Sam Houston.
Eastwood, Herbert K., (Mil.),
Sch. of Avn. Med., Randolph AFB.
Ellingson, Harold V., (Mil.),
Hdqrs., 3790th S.C. Gp., Gunter AFB, Ala.
Ellington, Jesse C.,
331 W. Nueva St., San Antonio.
Ellis, Sam, 710 Med. Arts Bldg., San Antonio.
Elmendorf, Hugo F., Jr.,
215 Camden, San Antonio.
Estrada, Felipe,
1216 Nix Prof. Bldg., San Antonio.
Estrada, Ramiro P.,
318 N. Santa Rosa, San Antonio.
Fairweather, M. Jeanne,
414 Corona, San Antonio.
Farinacci, Charles J., (Mil.),
121 Five Oaks Dr., San Antonio.
Farrell, Elnora Anne,
2819 N. McCullough, San Antonio.
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216 Med. Prof. Bldg., San Antonio.
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Fetzer, William J.,
1530 W. Summit, San Antonio.
Finney, James W.,
200 Donaldson, San Antonio.
Finsterwald, James F.,
119 General Krueger, San Antonio.
Fischer, Albert,
611 Med. Prof. Bldg., San Antonio.
Fisher, Rowan E.,
1109 S. Texas Bldg., San Antonio.
Flaherty, Bernard E., (Mil.),
1171 Monticello Rd., Lafayette, Calif.
Flinn, Don E., (Mil.),
Sch. of Avn. Med., Randolph AFB.
Folbre, Thomas W.,
1219 Nix Prof. Bldg., San Antonio.
Fox, Morris E., 307 Dwyer St., San Antonio.
Franke, Winthrop I.,
2608 N. Main Ave., San Antonio.
French, Jack A.,
906 Fredericksburg, San Antonio.
Galindo, Desiderio L.,
7038 Callaghan Rd., San Antonio.
Galloway, Ballard E.,
306 Houston Bldg., San Antonio.
Garnett, Walter L.,
Paseo Reforma 510, Mexico 6, D. F., Mex.
Garza, Remberto,
315 N. San Saba, San Antonio.
Geissler, Wallace H.,
313 Med. Prof. Bldg., San Antonio.
Geppert, Leo J., (Mil.),
Brooke Army Hosp., Ft. Sam Houston.
Gerodetti, Orlando F.,
344 Mary Louise Dr., San Antonio.
Giesecke, Carl G., (Mil.),
457 Graham, San Antonio.
Glass, Thomas G., Jr.,
102 Med. Prof. Bldg., San Antonio.
Goeth, Carl F.,
603 Med. Prof. Bldg., San Antonio.
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1133 Med. Arts Bldg., San Antonio.
Gonzalez, Hesiquio N.,
318 N. Santa Rosa, San Antonio.
Gonzalez, Joaquin B.,
318 N. Santa Rosa, San Antonio.
Goodnight, James E.,
1005 Nix Prof. Bldg., San Antonio.
Goodpasture, John E.,
2407 W. Huisache, San Antonio.
Gootee, Hubert George,
4617 Hummingbird, Houston.
Gordon, Marie D.,
205 Camden St., San Antonio.
Gordon, Wm. H., Jr.,
205 Camden St., San Antonio.
Gorsuch, Paul L.,
1208 Nix Prof. Bldg., San Antonio.
Gossett, Robert F.,
205 Camden St., San Antonio.
Grant, Harold,
602 W. French Pl., San Antonio.
Grantham, Edwin S.,
124 Dallas St., San Antonio.
Graves, William E.,
804 W. Poplar St., San Antonio.
Gray, Arthur M., 119 Burkedale, San Antonio.
Greer, Sam J., Jr.,
823 Nix Prof. Bldg., San Antonio.
Gregory, Ernest J., Jr.,
Box 7206, Hackberry Sta., San Antonio.
Gregory, Eugene B.,
1131 S. W. W. White Rd., San Antonio.
Griffin, Robert W.,
1432 Nix Prof. Bldg., San Antonio.
Grissom, Paul M., (Mil.),
USAF Hosp., Lackland AFB.
Griswold, James A.,
3506 S. New Braunfels, San Antonio.
Haggard, Charles H.,
1228 Med. Arts Bldg., San Antonio.
Haggard, Frank N.,
1228 Med. Arts Bldg., San Antonio.
Haley, Robert Roscoe,
216 N. Santa Rosa, San Antonio.
Hall, Augustus A.,
222 Med. Prof. Bldg., San Antonio.
Hardy, Robert C.,
922 Nix Bldg., San Antonio.
Hargis, W. Huard, Sr., (Hon.),
205 Camden St., San Antonio.
Harle, Raymond P.,
6936 San Pedro, San Antonio.
Hartman, Albert W., Jr.,
809 Nix Prof. Bldg., San Antonio.
Hartman, Henry C., (Hon.),
1104 Rigsby Ave., San Antonio.
Hartman, Ralph F.,
242 E. Beverly Mae, San Antonio.
Hausman, Robert,
Bexar Co. Court House, San Antonio.
Heaney, John P.,
1608 Nix Bldg., San Antonio.
Hebert, Thomas, 5305 So. Flores, San Antonio.
Heck, William H.,
1021 Nix Prof. Bldg., San Antonio.
Hekhuis, Gerrit L., (Mil.),
Aerospace Med. Cen., Brooks AFB.
Held, Edward C.,
114 Glenview Dr. W., San Antonio.
Helfer, Lewis M.,
208 Med. Prof. Bldg., San Antonio.
Henning, Garold G.,
110 Epworth, San Antonio.
Henry, Colvern D.,
416 Med. Prof. Bldg., San Antonio.
Henry, Mary M.,
601 Med. Arts Bldg., San Antonio.
Heny, Joseph S.,
307 N. Leona St., San Antonio.
Herff, Augustus F., Jr.,
1614 Nix Prof. Bldg., San Antonio.
Herff, Augustus F.,
1606 Nix Prof. Bldg., San Antonio.
Herff, Ferdinand P., (Hon.),
Box 155, San Antonio.
Hicks, Yale,
274 E. Edgewood Pl., San Antonio.
Hill, Alfred H.,
1216 S. Texas Bldg., San Antonio.
Hill, Lucius D., Jr.,
327 Med. Arts Bldg., San Antonio.
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809 Nix Prof. Bldg., San Antonio.
Hinchey, John J.,
1108 Nix Prof. Bldg., San Antonio.
Hogan, Thomas F., Jr.,
708 Nix Prof. Bldg., San Antonio.
Holcomb, Thomas M., (Mil.),
Lackland AF Hosp, San Antonio.
Hollan, O. Roger,
1110 S. Texas Bldg., San Antonio.
Holshouser, Chas. A.,
310 Med. Arts Bldg., San Antonio.
Hooper, Charles H., 605 Belknap, San Antonio.
Horner, Bernard G.,
3506 S. New Braunfels, San Antonio.
Hoskins, Henry R.,
514 Med. Arts Bldg., San Antonio.
Howard, David S.,
4119 Fredericksburg, San Antonio.
Howerton, Ernest E.,
201 Med. Prof. Bldg., San Antonio.
Hulse, Charles A.,
405 Med. Prof. Bldg., San Antonio.
Huntington, Ione,
519 Med. Prof. Bldg., San Antonio.
Ihrig, John J., (Mil.),
414 Quentin Dr., San Antonio.
Jackson, Carmault B., Jr., (Mil.),
3700th USAF Hosp., Lackland AFB.
Jackson, Dudley, Jr.,
1525 Nix Prof. Bldg., San Antonio.
Jackson, L. Walford,
906 Fredericksburg, San Antonio.
Jackson, Martha B., (Inac.),
410 Woodcrest Dr., San Antonio.
Jacob, Norman H.,
120 W. Elsmere, San Antonio.
Jacobs, Milton S.,
510 Med. Prof. Bldg., San Antonio.
Jensen, Andrew M.,
851 Avant Ave., San Antonio.
Jensen, Martin H.,
115 N. Park Blvd., San Antonio.
Jenson, Robert L.,
446 Canterbury Hill, San Antonio.
Johns, Sylvia M.,
414 Navarro St., San Antonio.
Johnson, Charles W.,
2121 Fredericksburg, San Antonio.
Johnson, Harry McC., Jr.,
1620 Nix Prof. Bldg., San Antonio.
Johnson, Max E.,
1220 Med. Arts Bldg., San Antonio.
Johnson, Robert L., (Mil.),
Sch. of Avn. Med., Randolph AFB.
Johnson, Ted, 106 Produce Row, San Antonio.
Johnson, William J.,
7407 Broadway, San Antonio.
Jones, Dean B.,
730 Med. Arts Bldg., San Antonio.
Jones, George W.,
929 Manor Dr., San Antonio.

Jones, L. Bonham,
929 Manor Dr., San Antonio.
Jordan, William S., (Mil.),
219 Fairchild, San Antonio.
Kaliski, Sidney R.,
402 Fulton Ave., San Antonio.
Kass, Albert,
705 Med. Arts Bldg., San Antonio.
Keedy, David M.,
1216 S. Texas Bldg., San Antonio.
Keil, Philip G., (Mil.),
410 Tyndall St., San Antonio.
Kelly, Roy J., (Mil.),
Sch. of Avn. Med., Brooks AFB.
Kenney, Nat M.,
222 E. Poplar St., San Antonio.
King, Billy D., 412 Ridgemont, San Antonio.
King, Dana G., Jr., (Mil.),
USAF Hosp., Lackland AFB.
King, Thomas C.,
1104 S. Texas Bldg., San Antonio.
King, W. A., (Hon.), Pandora.
Kitowski, Vincent J.,
5122 Hummingbird, Houston.
Klecka, Theodore A.,
214 Med. Prof. Bldg., San Antonio.
Kline, Philip S.,
619 Med. Prof. Bldg., San Antonio.
Klinger, Paul E.,
517 Med. Prof. Bldg., San Antonio.
Koch, Alvis A., 5148 Broadway, San Antonio.
Kohler, Colette M.,
328 Lamont Ave., San Antonio.
Koontz, Lee A., 205 Camden St., San Antonio.
Kopecky, Joseph, 1414 Nix Bldg., San Antonio.
Kopecky, Joseph Willis,
1408 Nix Prof. Bldg., San Antonio.
Kopecky, Leon C.,
1414 Nix Bldg., San Antonio.
Kost, Louis B.,
521 Med. Prof. Bldg., San Antonio.
Kraus, Ralph N., (Mil.),
Sch. of Avn. Med., Randolph AFB.
Krause, Robert B.,
518 W. Houston St., San Antonio.
Kupper, Roland C.,
114 El Prado Dr., San Antonio.
Kurtz, Irwin,
3104 W. Woodlawn, San Antonio.
Ladd, Graham B.,
424 Moore Bldg., San Antonio.
Lahourcade, Fred G.,
906 Fredericksburg, San Antonio.
Lamb, Lawrence E.,
415 Timberlane Dr., San Antonio.
Lambrecht, Daniel E.,
303 Wyndale, San Antonio.
Lampe, Juliet H.,
Spanish Oaks Tr. Pk., San Antonio.
Lampert, Morris H.,
1048 Hammond St., San Antonio.
Lancaster, Howard E., Jr., (Int.),
516 Lexington Ave., San Antonio.
Langdon, David E., (Mil.),
Sch. of Avn. Med., Brooks AFB.
Langlinais, Richard P.,
2819 N. McCullough, San Antonio.
Langner, C. Dwight,
440 Sheraton Dr., San Antonio.
Lea, Royal B.,
323 E. Melrose Dr., San Antonio.
Lee, Jack B., 409 Camden St., San Antonio.
Lehmann, C. Ferd,
218 Med. Prof. Bldg., San Antonio.
Leopold, Henry N.,
602 W. French Pl., San Antonio.
LePere, Robert H.,
1424 Nix Bldg., San Antonio.
Leslie, James T., Jr., (Mil.),
Lackland AF Hosp., San Antonio.
Lett, James E., (Mil.),
USAF Hosp., Lackland AFB.
Letteer, C. Ralph, Jr.,
933 Nix Prof. Bldg., San Antonio.
Levine, Bernard R.,
1625 Nix Prof. Bldg., San Antonio.
Levine, Robert, (Mil.),
USAF Hosp., San Antonio.
Lewis, Wade H., (Int.),
P. O. Box 96, Austin.
Livingston, Edward N., Box 1840, San Antonio.
Lowry, James K.,
Santa Rosa Hosp., San Antonio.
Luedemann, Waldo S.,
820 Nix Prof. Bldg., San Antonio.
Lundgren, Rupert W.,
1609 Nix Prof. Bldg., San Antonio.
Lyons, Robert E.,
110 W. El Prado, San Antonio.
Manhoff, Charles M.,
523 E. Quincy, San Antonio.
Manhoff, Louis J., (Hon.),
818 W. Woodlawn, San Antonio.
Manhoff, Louis J., Jr.,
1400 N. McCullough, San Antonio.
Mani, George C.,
745 W. Houston, San Antonio.
Mantell, Louis K., (Mil.),
817 Canterbury Hill, San Antonio.
Markette, Billy B.,
1400 N. McCullough, San Antonio.
Marshall, Joseph T.,
414 Robinhood Pl., San Antonio.
Martin, Frank M.,
403 W. Summit, San Antonio.
Martin, Richard L., (Mil.),
Lackland AF Hosp, San Antonio.
Martinez, Joseph J.,
537 Moore Bldg., San Antonio.
Masters, Robert A.,
1723 Buena Vista, San Antonio.
Matthaei, Pearl V.,
315 Rockhill Dr., San Antonio.
Matthews, John L.,
824 Nix Prof. Bldg., San Antonio.
Maurer, Robert T.,
1926 E. Lawndale Dr., San Antonio.
Maxwell, Ernest A.,
124 Dallas St., San Antonio.
Maxwell, W. Wortham,
626 Med. Arts Bldg., San Antonio.
May, Lester M.,
705 Med. Arts Bldg., San Antonio.
McCabe, Edward P., Jr.,
628 Med. Arts Bldg., San Antonio.
McComb, Asher R.,
1022 Nix Prof. Bldg., San Antonio.
McCullough, David,
1214 Nix Prof. Bldg., San Antonio.
McCurdy, Marion W.,
1034 Nix Prof. Bldg., San Antonio.
McGehee, Charles L.,
605 Med. Arts Bldg., San Antonio.
McIntosh, John A., (Hon.),
208 W. Woodlawn Ave., San Antonio.
McKay, Patrick H.,
628 Med. Arts Bldg., San Antonio.
McKiski, Wendell E.,
720 Tuxedo, San Antonio.
McMahon, David T., Jr.,
901 Med. Arts Bldg., San Antonio.
McMillan, Orin P.,
1120 S. Texas Bldg., San Antonio.
Meadows, John C., Jr.,
502 Med. Prof. Bldg., San Antonio.
Melenyzer, Charles L.,
511 New Moore Bldg., San Antonio.
Melton, Joseph T., (Mil.),
619 Fairchild, San Antonio.
Mena, A. I., 103 Buena Vista, San Antonio.
Mendez, Enrique, Jr., (Mil.),
Brooke Army Hosp., Ft. Sam Houston.
Metzner, Wesley R. T.,
534 Dwight Ave., San Antonio.
Meyer, Gerhard A.,
1734 Nix Prof. Bldg., San Antonio.
Milburn, Graham B.,
1104 Nix Bldg., San Antonio.
Miller, John B., Jr.,
1704 E. Commerce St., San Antonio.
Miller, Ralph B.,
Box 8335, Laurel Hts., San Antonio.
Miller, Robert A.,
1525 Nix Bldg., San Antonio.
Mims, James L., Jr.,
211 Med. Arts Bldg., San Antonio.
Miniel, Pedro R.,
315 N. San Saba, San Antonio.
Minter, Merton M.,
1734 Nix Prof. Bldg., San Antonio.
Mohle, Chester L.,
925 Contour Drive, San Antonio.
Monsalvo, Rudolph O.,
502 New Moore Bldg., San Antonio.
Montano, Ricardo G.,
603 W. Kings Hwy., San Antonio.
Montgomery, William D.,
511 W. French Pl., San Antonio.
Moore, George B., Jr.,
322 Med. Arts Bldg., San Antonio.
Moore, John M.,
818 Med. Arts Bldg., San Antonio.
Moore, Oliver S., (Hon.),
1846 W. Magnolia, San Antonio.
Moore, S. Foster, Jr.,
401 W. Summit, San Antonio.
Morris, Marion H.,
804 Nix Prof. Bldg., San Antonio.
Morrison, James B.,
1734 Nix Bldg., San Antonio.
Morron, Clyde R.,
1105 E. Commerce, San Antonio.
Moses, Louis E.,
414 Med. Prof. Bldg., San Antonio.
Mueller, Edwin L.,
1616 San Pedro, San Antonio.
Mueller, Edwin L., Jr.,
1616 San Pedro, San Antonio.
Mullen, Brooks W.,
1215 S. Texas Bldg., San Antonio.
Munslow, Ralph A.,
1233 Nix Prof. Bldg., San Antonio.
Murray, Neville,
827 Nix Prof. Bldg., San Antonio.
Nasta, Armando,
2608 N. Main Ave., San Antonio.
Nau, Cornelius H.,
351 E. Hildebrand, San Antonio.
Newton, Jerry, 3306 Roselawn, San Antonio.
Nichol, William W., (Mil.),
Brooke Army Hosp., Ft. Sam Houston.
Nicholson, John R.,
508 Med. Arts Bldg., San Antonio.
Nisbet, Alfred A.,
700 S. McCullough, San Antonio.
Nitschke, Richard E.,
5307 Broadway, San Antonio.
Nixon, James W.,
1121 Nix Prof. Bldg., San Antonio.
Nixon, James W., Jr.,
1121 Nix Prof. Bldg., San Antonio.
Nixon, Pat I., (Emer.),
523 Med. Prof. Bldg., San Antonio.
Nixon, Pat. I., Jr.,
523 Med. Prof. Bldg., San Antonio.
Nixon, Robert R.,
401 Med. Prof. Bldg., San Antonio.
Norman, Ruskin C.,
1502 Nix Prof. Bldg., San Antonio.
Novak, Lumir F.,
St. Anthony Hotel, San Antonio.
Novoa, Enrique,
103½ Buena Vista, San Antonio.
Oglesby, Paul C.,
711 Kirk Place, San Antonio.
Oliver, David R.,
1605 Nix Prof. Bldg., San Antonio.
O'Neill, Francis E.,
120 Med. Prof. Bldg., San Antonio.
O'Neill, James R.,
602 W. French Pl., San Antonio.
Ord, John W., (Mil.),
USAF Hospital, Lackland AFB.
Orlando, Anthony M.,
1723 Buena Vista, San Antonio.
Otero, Pedro J.,
413 International Bldg., San Antonio.
Owens, Ross, 807 Gibbs Bldg., San Antonio.
Oxford, M. Bradfield,
1602 Nix Bldg., San Antonio.
Palmer, Joseph W.,
7407 Broadway, San Antonio.
Park, J. Walter, III,
Nix Prof. Bldg., San Antonio.
Parker, Gerald W., (Mil.),
1322 Regent St., San Antonio.
Parrish, Robert E., (dead), San Antonio.
Parsons, John C.,
1125 Nix Prof. Bldg., San Antonio.
Partain, Jack M.,
205 Camden St., San Antonio.
Paschal, Frank L.,
1222 Nix Prof. Bldg., San Antonio.
Passmore, Ben H.,
1732 Nix Prof. Bldg., San Antonio.
Passmore, Glenn G.,
1529 Nix Prof. Bldg., San Antonio.
Payte, James T., 107 N. Cibolo, San Antonio.
Peterson, Robert C.,
510 Harrison Ave., San Antonio.
Peterson, Walter L.,
Box 7206, Hackberry Sta., San Antonio.
Pfeiffer, John P., Jr.,
1306 Oblate Dr., San Antonio.
Phillips, Claude M.,
444 Hammond Ave., San Antonio.
Phillips, Warren M.,
152 New Laredo Hwy., San Antonio.
Pipes, William F.,
2814 Hitching Post Rd., San Antonio.
Pipkin, J. Lewis,
714 Med. Arts Bldg., San Antonio.
Polka, James B.,
312 Med. Prof. Bldg., San Antonio.
Pomerantz, R. Bernard,
6936 San Pedro, San Antonio.
Posey, Frank M., Jr.,
101 McCullough Ave., San Antonio.
Post, S. Perry, 929 Manor Dr., San Antonio.
Poth, Duncan O.,
1230 Nix Prof. Bldg., San Antonio.
Potthast, Otto J.,
501 Gibbs Bldg., San Antonio.
Preacher, Madison L.,
916 E. Commerce, San Antonio.
Pressly, Thomas A.,
205 Camden St., San Antonio.
Price, Richard D.,
1233 Nix Bldg., San Antonio.
Pridgen, James E.,
424 Med. Prof. Bldg., San Antonio.
Pridgen, John L.,
908 Med. Arts Bldg., San Antonio.

Pryor, Jessie W.,
500 S.W. 24th St., San Antonio.
Pyterek, Arthur B.,
723 Med. Arts Bldg., San Antonio.
Rabel, John E.,
902 Nix Prof. Bldg., San Antonio.
Ragne, Edmund F.,
Box 7206, Hackberry Sta., San Antonio.
Rath, Albert E.,
1651 W. Mulberry, San Antonio.
Reeves, George D.,
202 Medford Dr., San Antonio.
Reily, William A.,
407 Med. Arts Bldg., San Antonio.
Reiter, Charles, Jr.,
1617 Nix Prof. Bldg., San Antonio.
Reppert, Lawrence B.,
Nix Prof. Bldg., San Antonio.
Ressmann, Arthur C.,
714 Med. Arts Bldg., San Antonio.
Reuter, Ernest G.,
611 Med. Arts Bldg., San Antonio.
Reveley, Hugh P.,
V. A. Center, Kecoughtan, Va.
Reveley, James E. L.,
2915 S. Presa St., San Antonio.
Rice, George W., (Inac.),
126 Medford Dr., S.E., San Antonio.
Richmond, Albert M.,
Nix Prof. Bldg., San Antonio.
Richmond, Harry A., Jr.,
608 Med. Prof. Bldg., San Antonio.
Richmond, James T.,
241 Donaldson Ave., San Antonio.
Richter, James K.,
3721 S. Presa, San Antonio.
Rinn, Odville A.,
810 Med. Arts Bldg., San Antonio.
Ritch, Allen T.,
1717 Nix Prof. Bldg., San Antonio.
Ritvo, Edward R.,
3707 Broadway, San Antonio.
Roan, Omer,
1222 Nix Prof. Bldg., San Antonio.
Robertson, Wilber F.,
511 Med. Prof. Bldg., San Antonio.
Rogers, Albert M.,
313 Med. Prof. Bldg., San Antonio.
Romo, Thomas, Jr.,
1706 E. Commerce St., San Antonio.
Rose, Howard N., (Mil.), Randolph AFB.
Rosenstein, Daniel L.,
Box 4038, Station A, San Antonio.
Rosenzweig, Milton M.,
1001 Med. Arts Bldg., San Antonio.
Rouse, J. W. H.,
2621 N. Main Ave., San Antonio.
Rubinstein, Barney, 258 Serenade, San Antonio.
Russ, Witten B., (Emer.),
205 Camden St., San Antonio.
Russell, Dan A., Jr.,
429 Med. Arts Bldg., San Antonio.
Sacks, David R.,
206 Med. Prof. Bldg., San Antonio.
Saegert, August H.,
Med. Arts Bldg., San Antonio.
Saenz, Daniel, 1723 Buena Vista, San Antonio.
Sallee, William T., (Mil.),
Sch. of Avn. Med., Randolph AFB.
Sammis, William L.,
325 Tuttle Road, San Antonio.
Samsel, Theodore B., Jr.,
504 Med. Prof. Bldg., San Antonio.
Sandler, Arthur S.,
8503 Wakefield, San Antonio.
Santa Cruz, Edgar W.,
401 W. Summit, San Antonio.
Sawtelle, William W.,
405 Med. Prof. Bldg., San Antonio.
Schattenberg, Herbert J.,
222 Med. Arts Bldg., San Antonio.
Schauer, Charles W.,
810 Med. Arts Bldg., San Antonio.
Schiffer, Sydney,
617 Med. Prof. Bldg., San Antonio.
Schwarting, Bland H.,
304 Med. Prof. Bldg., San Antonio.
Schwartzberg, Sam,
South Texas Bldg., San Antonio.
Scull, T. Jackson,
1005 Nix Bldg., San Antonio.
Severance, Alvin O.,
205 Camden St., San Antonio.
Shaeffer, Joseph R.,
745 W. Houston St., San Antonio.
Sharp, Thomas H.,
316 Med. Prof. Bldg., San Antonio.
Shaver, Benjamin B.,
211 Med. Prof. Bldg., San Antonio.
Shaw, Thad, (Inac.),
Gunter Hotel, San Antonio.
Shefts, Lawrence M.,
209 Paseo Encinal, San Antonio.
Shepherd, Virgil J., (Mil.),
4623 Hershey Dr., San Antonio.
Shepherd, Walter F.,
1401 Highland, San Antonio.
Shingle, Robert C.,
104 St. Cloud, San Antonio.
Shipman, E. D., (Inac.),
551 Cincinnati, San Antonio.
Shotts, Chester C.,
915 Med. Arts Bldg., San Antonio.
Sichi, William T.,
420 Med. Prof. Bldg., San Antonio.
Siever, James M.,
318 Med. Prof. Bldg., San Antonio.
Simpson, John W., Box 4038, San Antonio.
Skinner, Ira C., 124 Dallas St., San Antonio.
Skinner, Robert B., (Mil.),
167 Artillery Loop, San Antonio.
Skripka, Charles F.,
3721 S. Presa St., San Antonio.
Slayter, James E., Box 1840, San Antonio.
Smith, Forrest M., Jr.,
227 E. Hildebrand, San Antonio.
Smith, John M., Jr.,
110 El Prado Dr., W., San Antonio.
Smith, Thomas B., 102 Congress, San Antonio.
Smyth, Rodger G.,
331 W. Nueva St., San Antonio.
Snip, Russell T., 505 Howard, San Antonio.
Soma, Yone, 102 S. Rosillo, San Antonio.
Spann, Franklin L., (Mil.),
Brooke Army Hosp., Ft. Sam Houston.
Sparks, John E., (dead), San Antonio.
Spector, Morris,
906 Fredericksburg, San Antonio.
Stain, Stanley,
323 N. New Braunfels, San Antonio.
Standley, Eugene T.,
Med. Prof. Bldg., San Antonio.
Stanley, Ray W., 306 Patricia, San Antonio.
Stansell, Paul Q.,
2446 W. Summit, San Antonio.
Stanton, William P.,
1031 Nix Prof. Bldg., San Antonio.
Steed, P. Franklin,
1525 Nix Prof. Bldg., San Antonio.
Steinberg, Fred W.,
1525 Nix Prof. Bldg., San Antonio.
Stewart, C. Scott,
123 Ogden Lane, San Antonio.
Stieler, Albert,
1006 Med. Arts Bldg., San Antonio.
Stonehill, Robert B., (Mil.),
432 Fairchild, San Antonio.
Stovall, Virginia S., (Inac.),
606 Med. Prof. Bldg., San Antonio.
Strauch, James H.,
610 Med. Prof. Bldg., San Antonio.
Strozier, William E.,
2819 McCullough Ave., San Antonio.
Stubbs, F. Spencer,
114 W. Glenview Dr., San Antonio.
Stumpe, Alfred R., (Mil.),
Sch. of Avn. Med., Randolph AFB.
Sullivan, Thomas P.,
408 Med. Prof. Bldg., San Antonio.
Sutton, Robert S., 711 W. Kirk, San Antonio.
Sutton, Robert S., Sr., (Inac.),
711 W. Kirk, San Antonio.
Sweet, Horace C.,
205 Camden St., San Antonio.
Swinny, Boen,
314 Med. Arts Bldg., San Antonio.
Sykes, E. Meredith,
925 Nix Prof. Bldg., San Antonio.
Sykes, Edwin M., Jr.,
927 Nix Prof. Bldg., San Antonio.
Sykes, John H. J.,
201 Charles Rd., San Antonio.
Sylvester, Wendell R., (Int.),
515 Morales St., San Antonio.
Tarrow, Arthur B., (Mil.),
USAF Hosp., APO 633, New York, N.Y.
Taylor, Charles W., (Hon.),
276 W. Mandalay Dr., San Antonio.
Taylor, Thomas S.,
318 Med. Prof. Bldg., San Antonio.
Templeton, R. D., 133 Handley, San Antonio.
Tennison, Charles W.,
511 Med. Prof. Bldg., San Antonio.
Terrell, Robert W. B.,
316 Med. Prof. Bldg., San Antonio.
Thaddeus, Aloysius P.,
502 Med. Prof. Bldg., San Antonio.
Thaggard, Alvin, Jr.,
1129 Nix Bldg., San Antonio.
Thomas, Robert P., Jr.,
1002 Nix Prof. Bldg., San Antonio.
Thompson, Milton S.,
441 Burr Road, San Antonio.
Thomson, George W., Box 4216, San Antonio.
Thornton, Melvin L.,
351 E. Hildebrand, San Antonio.
Timmins, Oliver H., (Hon.),
1211 Med. Arts Bldg., San Antonio.
Timmins, Oliver H., Jr.,
1211 Med. Arts Bldg., San Antonio.
Tippit, Nathaniel G.,
Mayo Foundation, Rochester, Minn.
Todd, David A.,
1502 Nix Prof. Bldg., San Antonio.
Trevino, Saul S.,
318 N. Santa Rosa, San Antonio.
Trimmer, Kenneth E.,
205 Camden St., San Antonio.
Tucker, Lewis E.,
1402 Nix Bldg., San Antonio.
Tucker, Victor C.,
1402 Nix Prof. Bldg., San Antonio.
Ullrich, Fredric W., (Int.),
1720 Chapel Ct., Northbrook, Ill.
Urrutia, Adolfo,
210 N. Santa Rosa, San Antonio.
Urrutia, Carlos,
212 N. Santa Rosa, San Antonio.
Van Auken, Howard A.,
215 Camden St., San Antonio.
Veit, John P., (Mil.),
3810th USAF Hosp., Maxwell AFB, Ala.
Venable, Charles S., (Emer.),
154 Park Hill Dr., San Antonio.
Voltz, Phillip W., Jr.,
120 Med. Prof. Bldg., San Antonio.
Von Bose, Edda, 914 Kayton Ave., San Antonio.
Wait, Raymond B.,
929 Manor Dr., San Antonio.
Walker, Carl J.,
719 E. Houston, San Antonio.
Walker, Dorothy J. B.,
183 Thorain W., San Antonio.
Walker, H. Vincent,
Nix Prof. Bldg., San Antonio.
Waller, Edward P.,
205 Camden St., San Antonio.
Walling, Russell G.,
211 Richmond Ave., San Antonio.
Walsh, John N.,
508 Med. Prof. Bldg., San Antonio.
Walthall, Walter,
1114 S. Texas Bldg., San Antonio.
Walton, Lowell C., (Mil.),
USAF Hospital, Lackland AFB.
Ward, Mildred E.,
104 Babcock Rd., San Antonio.
Ware, Ray W., (Mil.),
Sch. of Avn. Med., Randolph AFB.
Watson, I. Newton,
906 Fredericksburg, San Antonio.
Watts, John A., (Hon.),
110 W. Lullwood, San Antonio.
Weatherford, Eddie W.,
205 Camden St., San Antonio.
Weatherford, Jack M.,
205 Camden St., San Antonio.
Webb, Hamilton B., (Mil.),
Sch. of Avn. Med., Randolph AFB.
Webb, John B., Jr., 205 Camden, San Antonio.
Weiner, Bernard K.,
929 Manor Dr., San Antonio.
Weiner, Myron F., (Mil.),
103 Pilgrim Dr., San Antonio.
Weiss, Victor J.,
509 Med. Arts. Bldg., San Antonio.
Weixel, Francis X.,
824 Nix Prof. Bldg., San Antonio.
Welch, Eldred E.,
503 New Moore Bldg., San Antonio.
Wells, Dolph T.,
3506 S. New Braunfels, San Antonio.
Welsh, Virginia C., 402 Fulton, San Antonio.
Wenger, Don S., (Mil.),
USAF Hospital, Lackland AFB.
Whittier, Charles A.,
928 E. Crockett, San Antonio.
Wier, Vernon S., 107 Eads Ave., San Antonio.
Wiesner, Jerome J.,
1128 Nix Prof. Bldg., San Antonio.
Wigodsky, Herman S.,
420 E. Houston, San Antonio.
Wilkinson, John M.,
307 Med. Arts Bldg., San Antonio.
Willerson, Eleanor T.,
108 Sheraton, San Antonio.
Willerson, J. E., (Hon.),
144 Barilla, San Antonio.
Willerson, Wm. D.,
1028 Nix Prof. Bldg., San Antonio.
Williams, Coyle W., Jr.,
1420 Nix Prof. Bldg., San Antonio.
Williams, Philip T., Jr.,
929 Manor Dr., San Antonio.
Wilson, Harry G.,
6612 S. Flores, San Antonio.
Wilson, William L.,
617 Med. Prof. Bldg., San Antonio.
Winter, John W.,
350 Terrell Rd., San Antonio.
Winter, William C., Jr., (Mil.),
USAF Hospital, San Antonio.
Woldman, Alvin L.,
929 Manor Dr., San Antonio.

Wolf, William M.,
1717 Nix Prof. Bldg., San Antonio.
Wong, Peter Y., 5617 Avenue R, Galveston.
Woodward, George S., (Mil.),
USAF Hosp., Lackland AFB.
Works, Bynum M., Jr.,
110 El Prado Dr., W., San Antonio.
Worsham, John W.,
1400 McCullough, San Antonio.
Wright, Jack M.,
403 W. Summit, San Antonio.
Wyatt, Byron W.,
603 Navarro St., San Antonio.
Ximenes, E. T.,
318 N. Santa Rosa, San Antonio.
Yuen, Lila, Box 7206, San Antonio.
Zaccaria, Attilio, Jr.,
603 Lockhill Selma, San Antonio.
Zeitlin, S. P., 6500 S. Flores, San Antonio.
Zellmer, Robert W., (Mil.),
Radiobiology Br., Brooks AFB.
Zink, Pearl L.,
615 Med. Arts Bldg., San Antonio.
Zuschlag, Ella,
611 Med. Prof. Bldg., San Antonio.

COMAL

Bergfeld, Arthur W. C.,
Box 49, New Braunfels.
Bergfeld, Jack A., Box 859, New Braunfels.
Frueholz, Bertha, Box 960, New Braunfels.
Frueholz, Fred, Box 960, New Braunfels.
Frueholz, Frederick, Jr.,
Box 960, New Braunfels.
Hinman, Alexander J., Box 79, New Braunfels.
Holtman, Harold A.,
255 San Antonio, New Braunfels.
Kennady, Donald S.,
532 N. Union St., New Braunfels.
Langston, John H., Jr.,
243 S. Seguin Ave., New Braunfels.
Schaefer, John K.,
P. O. Box 487, New Braunfels.
Schleicher, Leroy C.,
670 Marion Rd., New Braunfels.
Woodward, Stanley M.,
535 S. Seguin, New Braunfels.

GONZALES

Elder, Nathan A., (Hon.), Nixon.
Henkel, Cecil L., 318 St. Peter, Gonzales.
Kirkpatrick, Marjorie,
334 E. 25th St., New York, N. Y.
Nixon, Sam A., Jr., Box 548, Nixon.
Ponder, Stewart M., Waelder.
Price, James C., 521½ St. Joseph, Gonzales.
Shelby, David M., Gonzales.
Sievers, Walter A., Gonzales.
Stahl, Louis J., Box 184, Gonzales.
Von Werssowetz, Odon F.,
Warm Springs Foundation, Gonzales.

GUADALUPE

Bachman, George P., 110 W. Mountain, Seguin.
Douthett, J. C. B., (Hon.)
110A N. Austin, Seguin.
Goetz, Joseph T., 109 W. Gonzales, Seguin.
Heinen, Allen I., 1025 N. Austin, Seguin.
Knolle, Robt. L., Jr., 110 W. Mountain, Seguin.
Liberty, Herbert G., 104 E. Court St., Seguin.
Mays, Anthony W., Jr., (Mil.),
1025 N. Austin, Seguin.
Moore, Henry H., Jr., 105 N. River, Seguin.
Mueller, John A., 1025 N. Austin, Seguin.
Raetzsch, Andrew, 109 W. Gonzales, Seguin.
Randolph, Vivien P., (Hon.), Schertz.
Ray, Robert S., 105 N. River St., Seguin.
Weiss, Leroy E., 1025 N. Austin St., Seguin.
Williams, Jesse B., 110 W. Mountain, Seguin.

KARNES-WILSON

Blake, John V., Jr., Floresville.
Blaustone, Henry H., 223 Main St., Kenedy.
Bonnstetter, Harold J., Box 638, Kenedy.
Boykin, Solomon R., Floresville.
Day, Ancie Fred, Jr., P. O. Box 357, Runge.
Edwards, Thomas P., Karnes City.
Haverlah, Gene V., Poth.
Mills, William C., Jr., Kenedy.
Oxford, Jerry W., (Hon.), Floresville.
Quillian, Causey C., 330 W. Live Oak, Kenedy.
Shannon, Shelby E.,
P. O. Box 1098, Karnes City.

KERR-KENDALL-GILLESPIE-BANDERA

Andrews, Ernest C.,
204 E. Austin, Fredericksburg.
Bacon, Dan W.,
Peterson Hosp. Bldg., Kerrville.
Bell, William E., 313 Circle Dr., Kerrville.
Birt, John B., Harper.
Brandenstein, Luise C.,
Box 907, Legion Br., Kerrville.
Brown, Dor W., Jr.,
109 S. Adams, Fredericksburg.
Burgess, Richard M., Jr., Box 711, Kerrville.
Byrd, William C., Jr.,
325 Peterson Hosp., Kerrville.
Day, Harold C., 31 Main St., Boerne.
Drane, Hugh A., Jr., Kerrville.
Dyer, Edward L., Kerrville.
Eley, Julia S., Kerrville.
Feller, Lorence W., Fredericksburg.
Gregg, William E., 607 W. Water, Kerrville.
Gremmel, Gilbert C., P. O. Box 428, Boerne.
Guill, Russell E., Sid Peterson Hosp., Kerrville.
Haffner, Vorha M. B., Box 163, Kerrville.
Harzke, Otto F., Comfort.
Jackson, J. Warren, V. A. Hospital, Kerrville.
Jones, Chas. C., Jr., 720 W. Main, Kerrville.
Jones, Charles C., Sr., (Hon.), Comfort.
Knapp, Dwight R.,
317 Peterson Hosp., Kerrville.
Matthews, Choice B.,
305 Peterson Hosp., Kerrville.
Meador, George D.,
Bandera Hosp. Bldg., Bandera.
Monroe, Myrick L., Box 375, Kerrville.
Neigut, Joseph S., 209 N. 10th, Junction.
Packard, Duan E., Kerrville.
Perry, J. Hardin, Fredericksburg.
Redland, Arthur J.,
421 Westminster St., Kerrville.
Ross, Luther W., P. O. Box 711, Kerrville.
Rountree, James T., T.B. Hosp., Kerrville.
Seidel, Joshua, 1104 W. Main, Kerrville.
Simpson, Robt. Keith, (Hon.), Kerrville.
Springall, Walton H., Fredericksburg.
Stein, Edward F., Jr.,
258 E. Main St., Fredericksburg.
Turner, Carl K., 209 N. 10th St., Junction.
Williams, Barney K., Jr., Box 872, Kerrville.

LaSALLE-FRIO-DIMMIT

Barnard, W. L., (Hon.), Carrizo Springs.
Brown, Dorothy V. M., Box 53, Catarina.
Crawford, John M., Carrizo Springs.
Lindley, Calvin D., (Hon.), (dead),
Carrizo Springs.
McKinley, Robert L., (Mil.), Pearsall.
Myers, Clyde P., Cotulla.
O'Connor, Timothy D., Pearsall.
Pickett, Jack B. E., Jr., (Int.),
V. A. Hospital, Hines, Ill.
Pickett, B. E., Sr., Carrizo Springs.
Primomo, John S., Dilley.
Primomo, Marion P., Dilley.
Wilson, Donald K., Box 55, Carrizo Springs.
Wilson, Emmett N., Jr., Box 696, Pearsall.

MEDINA-UVALDE-MAVERICK-
VAL VERDE-EDWARDS-REAL-KINNEY-
TERRELL-ZAVALA

Allison, George M., 126 W. Nopal St., Uvalde.
Brittain, Mary Ruth,
204 E. Zavala, Crystal City.
Burditt, Bucky L., 901 Griner, Del Rio.
Burgess, George A.,
248 Jefferson St., Eagle Pass.
Calderon, Fermin, 838 S. Main St., Del Rio.
Cartall, Louis M., 300 Dignowity St., Del Rio.
Cunningham, George B.,
126 W. Nopal St., Uvalde.
Dimmitt, Dean P., Uvalde.
Donaldson, Elizabeth, Ross Bldg., Del Rio.
Eads, Ray A., Uvalde.
Eckert, Robert R.,
Cast. Hosp., and Clin., Castroville.
Fly, Sterling H., Jr.,
105 N. Piper Lane, Uvalde.
Folsom, Charles W.,
14557 Temple Ave., La Puente, Calif.
Gates, Ellis F., P. O. Box 474, Eagle Pass.
Graham, Robert N., Del Rio.
Guice, Leroy E., 231 S. Getty, Uvalde.
Herrmann, George H.,
301 Del Rio Bank Bldg., Del Rio.
Hyslop, James R., V. A. Hosp., McKinney.
Johnson, Thomas M.,
300 Dignowity St., Del Rio.
Kaback, Harry, 421 Washington St., Eagle Pass.
LaForge, Hershall, Uvalde.
Lewis, B. Oliver, P. O. Box K, Eagle Pass.
McWilliams, William R.,
Del Rio Natl. Bank Bldg., Del Rio.
Meredith, William P., Del Rio.
Merritt, George H., (Hon.),
Rocksprings Road, Uvalde.
Meyer, Walter B., Hondo.
Mims, Joe M., (Int.),
Robt. B. Green Hosp., San Antonio.
Montemayor, Raul M.,
333 Ceylon St., Eagle Pass.
Newby, Hi E., 300 W. Nignowity, Del Rio.
Peters, Leo E., Devine.
Poindexter, Cary A., Crystal City.
Pratt, Frank H., Rocksprings.
Rankin, Richard C., Jr.,
121 W. South St., Uvalde.
Rodriguez, Simon, 1405 S. Main, Del Rio.
Scott, Robert E., Zachary Bldg., Uvalde.
Spencer, John C., 212 Maverick, Crystal City.
Surber, Maxine J.,
701 Rocksprings Rd., Brackettville.
Sutton, Claud R., Jr., 126 W. Oak, Uvalde.
Turullols, Jesus, Sr., 901 Griner St., Del Rio.
Wurzbach, Fay, Castroville.

SIXTH DISTRICT

Dr. S. W. Bohmfalk, Weslaco, Councilor

BEE-LIVE OAK-McMULLEN

Adlof, Carolyn M., Box 206, Three Rivers.
Edmondson, John W.,
908 N. St. Marys, Beeville.
Lancaster, Howard E., Box 559, Beeville.
McNeill, Scott E., Beeville.
Miller, Ernest E., Beeville.
Muecke, Elmo W., Three Rivers.
Reagan, James L.,
310 N. Washington, Beeville.
Reagan, John W., 908 N. St. Marys, Beeville.
Reagan, Tom B., Beeville.
Sansom, George W., George West.

BROOKS-DUVAL-JIM WELLS

Albert, Richard O., 64 N. Wright St., Alice.
Allison, Albert M., Box 1254, Alice.
Buck, Haldor R., Premont.
Crisp, Euell W., Jr., 501 N. Reynolds St., Alice.
Davis, Glenn B., Box 576, Falfurrias.
Garcia, Jose A., 310 N. Wright St., Alice.
Glendenning, F. C., Hebbronville.
Gonzalez, Juan C., Benavides.
Haag, Edmund L., Jr., Freer.
Hocott, Joseph F., Freer.
Howard, Glenn T., Box 1277, Alice.
Joseph, Philip S., 1009 E. Sixth, Alice.
Moet, John Allen, Orange Grove.
Moore, Victor, Premont.
Newkirk, William H., 1031 E. Main St., Alice.
O'Neil, Andrew W., Falfurrias.
Penly, Richard S., Falfurrias.
Riddle, Riley N., 1009 E. 6th St., Alice.
Turnham, John C., Jr., 1009 E. Sixth St., Alice.
Virgin, Edwin P., 501 N. Reynolds, Alice.
Wilder, Lowell E., Falfurrias.
Williams, J. Harold, Alice.
Wyche, Geo. G., St., 310 N. Wright St., Alice.

CAMERON-WILLACY

Allen, George Earl, (Mil.), Harlingen.
Amidon, Vivien M., McKelvey Bldg., Harlingen.
Ashcraft, E. Jeff, Jr.,
613 W. Filmore, Harlingen.
Atchison, Martin V.,
1622 Ed Carey Dr., Harlingen.
Baden, Ervin E., 152 S. 6th St., Raymondville.
Beazley, H. Liston,
1716 Ed Carey Dr., Harlingen.
Bedri, Marcel R.,
501 McKelvey Bldg., Harlingen.
Benavides, Simon I., Jr.,
335 East Kimball, Raymondville.
Bennack, Gene E., Box 778, Raymondville.
Bennack, George E.,
152 S. 6th St., Raymondville.
Berrey, Bedford H., (Mil.),
108 Lyman Dr., San Antonio.
Binney, Charles, II,
1720 Ed Carey Dr., Harlingen.
Bleakney, Phil A., 214 E. Harrison, Harlingen.
Breeden, Roy F.,
105 W. Elizabeth, Brownsville.
Breyer, Amy, (Inac.),
1 Ebony Drive, Brownsville.
Brown, John F., Jr.,
315 N. Sam Houston, San Benito.
Caldeira, Frederick D.,
113 W. Van Buren, Harlingen.
Calderoni, Francisco F.,
37 W. Elizabeth, Brownsville.
Cannon, George M., (Int.),
1402 S. Kentwood, Springfield, Mo.
Carter, Samuel C., State T.B. Hosp., Harlingen.
Casey, James D.,
400 W. Highway 77, San Benito.
Cash, Clarence M., (Hon.),
205 S. Llano, Junction.
Chase, Ned B., Jr., (Mil.), San Benito.
Childress, James L.,
1710 Ed Carey Dr., Harlingen.

Clarke, James Y.,
2020 W. Charleston, Las Vegas, Nev.
Conlin, F. Dixon,
1619 Ed Carey Dr., Harlingen
Cope, Solomon F., 1120 Garcia, Port Isabel.
Cowgill, David M.,
1135 N. Sam Houston, San Benito.
Davis, Lum M., (Hon.),
709 E. Harrison, Harlingen.
De La Garza, Enrique, Box 2042, Brownsville.
Destefano, Frederick W.,
55 W. Elizabeth, Brownsville.
Dieck, John A., 211 E. Harrison, Harlingen.
Duncan, George W.,
1710 Ed Carey Dr., Harlingen.
Dyo, Kaoru, 324 E. Harrison St., Harlingen.
Englerth, Fred L., 306 S. 3rd St., Harlingen.
Ferris, John A., Jr., Box 778, La Feria.
Flory, David W., P. O. Box 271, Harlingen.
Freeman, George A.,
1155 Parkwood Pl., Brownsville.
Fryer, Ronald E.,
715 W. Jefferson, Brownsville.
Gallaher, George L.,
McKelvey Bldg., Harlingen.
Garza, Luis, 1601 South F, Harlingen.
George, James C., II,
705 W. Jefferson, Brownsville.
Goerger, Verne F.,
665 W. Riggs, Raymondville.
Griffey, Earle B.,
915 Palm Blvd., Brownsville.
Guerrero, Emilio,
1049 E. Washington, Brownsville.
Haas, Nelson W., Box 789, San Benito.
Hamilton, Oscar A.,
1626 Ed Carey Dr., Harlingen.
Harrop, L. Louis,
322 E. Monroe Ave., Harlingen.
Hartman, John T.,
1616 Ed Carey Dr., Harlingen.
Hawkins, Beatrice W.,
P. O. Box 1747, Brownsville.
Hawkins, W. W.,
105 W. Elizabeth St., Brownsville.
Hecht, Rudolph C., P. O. Box 686, La Feria.
Heins, Donald E.,
336 S. Eighth St., Raymondville.
Heins, Otto H., Raymondville.
Higgs, Paul C.,
212 Clarke & Cts., Harlingen.
Hockaday, James A., Port Isabel.
Jackson, Robert C.,
1614 Ed Carey Dr., Harlingen.
Jaros, Stanislaus H., 308 S. 3rd St., Harlingen.
Jondahl, Willis H., P. O. Box 1727, Harlingen.
Jones, Lee Dale,
525 Villa Maria Blvd., Brownsville.
Keeble, Leon, Jr.,
525 Villa Maria Blvd., Brownsville.
Kinder, Thurman A., Jr.,
248 E. Levee St., Brownsville.
Krishna, Ikbal,
47 W. Elizabeth St., Brownsville.
Kruisheer, H. E. J.,
817 E. Harrison, Harlingen.
Kuppinger, John C.,
613 W. Filmore, Harlingen.
Lamm, Annie T.,
1025 S. 16th St., Harlingen.
Lamm, Heinrich,
1025 S. 16th St., Harlingen.
LaMotte, Thomas J.,
613 W. Filmore, Harlingen.
Lancaster, David D., Box 393, San Benito.
Lawrence, Oscar V., (Hon.), (dead),
Brownsville.
Longoria, Vidal, 800 E. Adams, Brownsville.
Lowery, George S., Box 155, Rio Hondo.
Loyd, Edward M., Box 2061, Harlingen.
Lyle, Charles F., Box 996, San Benito.
Martin, A. G. M., III,
1616 Ed Carey Dr., Harlingen.
McLean, Edwin P.,
24 S. E. St. Charles, Brownsville.
Merrill, Samuel J.,
715 W. Jefferson, Brownsville.
Miller, Harry Allen, Jr.,
301 E. Washington, Brownsville.
Miller, John B.,
301 E. Washington, Brownsville.
Muniz, Leofredo G.,
115 E. Harrison, Harlingen.
Nickell, David F.,
322 E. Harrison, Harlingen.
Noell, L. Pope, Jr.,
1202 S. 15th St., Harlingen.
Olcott, Cornelius, Jr.,
218 E. Harrison, Harlingen.
O'Neal, Kermit C.,
1022 S. 16th St., Harlingen.
Packard, John P., 214 E. Harrison, Harlingen.
Palmer, Mahlon P.,
1314 E. Hwy 77, Harlingen.
Para, Andrew W.,
210 1st Natl. Bank, Brownsville.
Parker, S. Mitchell,
480 N. Sam Houston, San Benito.
Pearson, Bernard S., P. O. Box 390, Harlingen.
Petta, George H., P. O. Box 1727, Harlingen.
Poole, Pierre P., 44 East Levee, Brownsville.
Quintanilla, Carlos J., Box 592, Harlingen.
Reeder, John W., Jr.,
216 E. Harrison St., Harlingen.
Rodriguez, Hesiquio, Box 1790, Harlingen.
Ross, William F.,
315 N. Sam Houston, San Benito.
Roth, Karl A., 105 W. Elizabeth, Brownsville.
Salinger, Leland M.,
105 W. Elizabeth, Brownsville.
Scales, Hunter L., Jr.,
375 N. Sam Houston, San Benito.
Scanlan, Nestor,
450 W. Jefferson, Brownsville.
Shafer, Troy A., 561 Lake Drive, Harlingen.
Sherman, K. C., 300 S. 4th St., Harlingen.
Shultz, Donald E., Coronado Bldg., Harlingen.
Shultz, Norma P., 1314 E. Hwy. 77, Harlingen.
Simmons, Ray L., II,
915 Palm Blvd., Brownsville.
Smith, Dudley W.,
10 E. Elizabeth, Brownsville.
Smith, Robert N., Jr.,
1616 Ed Carey Dr., Harlingen.
Spence, Charles H., Jr.,
336 S. 8th, Raymondville.
Stephens, John M.,
P. O. Box 1733, Brownsville.
Tewell, Howard E., Jr.,
1716 Ed Carey Dr., Harlingen.
Thurman, George E.,
222 E. Harrison Ave., Harlingen.
Vogel, Martha E., Box 117, Santa Rosa.
Walsworth, Frank D.,
112½ S. First, Harlingen.
Watkins, John C., (Hon.),
Box 866, Harlingen.
Welty, John Allen,
1720 Ed Carey Dr., Harlingen.
Willeford, George,
1720 Ed Carey Dr., Harlingen.
Withers, John C.,
1154 E. Elizabeth, Brownsville.
Works, Bynum M.,
105 W. Elizabeth, Brownsville.
Works, Lee, 105 W. Elizabeth, Brownsville.
Youngberg, Stephen A., Box 404, Port Isabel.

HIDALGO-STARR

Blocker, William P., P. O. Box 887, Donna.
Bohannon, Richard F., 225 West Park, Pharr.
Bohmfalk, Stanley W.,
587 Kansas Ave., Weslaco.
Brandes, Gerald E., 817 Quince, McAllen.
Burnett, Thomas R., (dead), Mission.
Caldeira, Antonio D., 541 S. Texas, Mercedes.
Caldwell, James W.,
606 S. Broadway, McAllen.
Cantu, Carlos L., 126 E. Park St., Pharr.
Carleton, Thomas M., 914 Miller St., Donna.
Casso, Ramiro R., 620 S. Main St., McAllen.
Caston, McKee, 1607 N. 8th St., McAllen.
Conwell, Halford R., 526 Third St., Mercedes.
Cuellar, Armando,
101-A S. Kansas Ave., Weslaco.
DeWitt, Joseph L., 201 N. Broadway, Elsa.
Douglass, Daniel D., 412 S. Main, McAllen.
Duncan, Wallace H., 1411 Beaumont, McAllen.
Edwards, Thomas G., 301 Texas, Mercedes.
Ellis, Jack R., 427 Kansas Ave., Weslaco.
Evans, Sheridan S., 619 S. Broadway, McAllen.
Fitch, James F., 817 Quince, McAllen.
Forcher, Henry, 521 S. 12th St., Edinburg.
Frink, Berton F., M & J Nelson Bldg., McAllen.
Garcia, Octavio, 720 S. Main, McAllen.
Garcia, Raul, 620 S. Broadway, McAllen.
Garcia-Esteves, Juan, 820 Pecan, McAllen.
Garza, Rafael, 718 S. Main, McAllen.
Gilliam, Hilburn D.,
416 S. Broadway, McAllen.
Gordon, Clarence E., 412 S. Main St., McAllen.
Graham, Ronald A., 710 S. Cage, Pharr.
Greene, Maurice L., 821 Quince, McAllen.
Guerra, Gilbert A., 107 S. 13th St., Edinburg.
Guerra, Lauro G., 701 S. 16th St., McAllen.
Gustafson, Wesley A.,
1st Natl. Bank Bldg., McAllen.
Hamme, Ralph E., 505 S. Closner, Edinburg.
Harren, Henry F., Jr., 427 Kansas, Weslaco.
Hatfield, Walter H., Box 1598, McAllen.
Hoffmaster, Vance D.,
521 S. Closner, Edinburg.
Hough, Travers E., Jr.,
509 S. Closner, Edinburg.
Ice, Noel V., 1405 S. 53rd St., Temple.
Ivy, J. Bryan, 427 Kansas, Weslaco.
Johnston, Robert H., Mercedes.
Kellar, Robert J., 530 S. Texas, Weslaco.
Kuhl, Ivan W., 414½ S. Main, McAllen.
Lancaster, George M.,
587 Kansas Ave., Weslaco.
Lawler, Marion R., 418 S. Texas, Mercedes.
Lazo, Francisco G., 315 Missouri St., Weslaco.
Livengood, Gerald S., 1122 Conway, Mission.
Long, William H., 129 E. Caffery, Pharr.
Lovell, Barney K., 416 S. Broadway, McAllen.
Lubben, John F., Jr., 1414 Galveston, McAllen.
Marsh, Elinor E., 713 Vine, McAllen.
Martin, James C., Jr., 1122 Conway, Mission.
Maxwell, Walter J., Jr.,
817 Quince St., McAllen.
May, Joe W., 507 S. Closner, Edinburg.
McCalip, Edwin L., (Hon.), Weslaco.
McKinsey, S. Joe, 802 South Main, McAllen.
Mims, Charles H., 1301 Conway, Mission.
Mock, Duane V., 109 E. 7th, San Juan.
Munal, H. Deane, Box 66, San Juan.
Narro, George, Box 3457, McAllen.
Nordmeyer, Arthur F., 105 W. 9th St., Mission.
North, Norman T., 1008 Doherty, Mission.
Osborn, Frank E., (Hon.),
303 S. 10th St., McAllen.
Osborn, Robert W., Box 329, McAllen.
Palisano, Philip A., 221½ S. Main, McAllen.
Panzer, Ralph P., 704 S. Texas, Weslaco.
Queen, Charles R., 305 Ebony Lane, Edinburg.
Rabinowitz, George E., 820 Pecan St., McAllen.
Ramirez, Mario E., Roma.
Reed, George A., Box 741, Weslaco.
Reed, Walter Earl, 107 East 7th, San Juan.
Reidland, Kenneth E.,
503 S. Broadway, McAllen.
Rhea, Keith, Rt. 1, Box 276, McAllen.
Riley, Pat, 213 E. 10th St., Mission.
Roberts, Howard H., 418 S. Texas, Mercedes.
Rodriguez, M. J.,
308 E. 4th St., Rio Grande City.
Sanchez, Santiago A.,
Scott & White Clin., Temple.
Schutz, Joe D., 1414 Galveston St., McAllen.
Scott, Kincy J., 402 S. Cage, Pharr.
Shamburger, W. H., Jr.,
P. O. Box 308, San Juan.
Sherfy, Hilde V. W., 1812 S. 11th, McAllen.
Smith, Lloyd, 708 South Main, McAllen.
Southwick, Lloyd M., Box 398, Edinburg.
Sybilrud, Hjalmer W.,
620 S. Broadway, McAllen.
Terrell, Pruitt D.,
606 S. Broadway, McAllen.
Trevino, Jorge H., 718 S. Main St., McAllen.
Webb, John B., 104 N. Fourth St., Donna.
Whigham, Herschel E., 412 S. Main, McAllen.
Whigham, William E., (Hon.),
414 S. Broadway, McAllen.
Wooldridge, Dean H., 817 Quince, McAllen.
Young, Ray L., Box 307, Mission.

KLEBERG-KENEDY

Barnett, Lawrence M., Box 608, Bishop.
Boyd, Newell D.,
227 W. Kleberg St., Kingsville.
Dunn, S. Chester,
831 Alexander St., Kingsville.
Ewert, William A.,
704 E. Ceasar St., Kingsville.
Gaston, Earl, Box 1311, Kingsville.
Ginther, Clarke E., Box 508, Bishop.
Greif, Emmett W.,
613 E. Ragland St., Kingsville.
Higgins, Frank B.,
230 W. Yoakum St., Kingsville.
Jones, A. C., 730 W. Lee St., Kingsville.
Parnell, Claude J., Jr.,
928 S. 14th St., Kingsville.
Peace, Dewey W., Box 608, Bishop.
Peace, Dewey W., Jr., Box 608, Bishop.
Ramey, Lindell E., Box 431, Kingsville.
Scales, James R., Box 1311, Kingsville.
Sublett, Collier M., 723 W. Lee St., Kingsville.
Walling, Bert H., Jr.,
227 W. Kleberg, Kingsville.
Watson, John D., Jr.,
227 W. Kleberg, Kingsville.
Wiles, William T., (Hon.), Riviera.

NUECES

Abbey, Joseph A.,
2451 Morgan, Corpus Christi.
Ahern, Gerald S.,
1017 Santa Fe, Corpus Christi.
Alsop, Joseph F.,
3154 Reid Dr., Corpus Christi.
Anderson, William E.,
1001 Louisiana, Corpus Christi.
Appel, Myron H.,
1200 Santa Fe, Corpus Christi.
Arnim, Landon C.,
2424 Morgan, Corpus Christi.

Arnold, Harrell R.,
1214 Santa Fe, Corpus Christi.
Ashmore, Alvin J.,
1800 S. Staples Bldg., Corpus Christi.
Barnard, James L.,
1240 3rd St., Corpus Christi.
Barnard, Wm. C., 1240 3rd St., Corpus Christi.
Barnes, George B.,
2461 Morgan Ave., Corpus Christi.
Bernwanger, Damon C.,
3639 S. Staples, Corpus Christi.
Bickley, Estill T.,
2524 Morgan, Corpus Christi.
Blaine, Alva L., 2466 Morgan, Corpus Christi.
Blaine, Mose H., Jr.,
2466 Morgan, Corpus Christi.
Blair, John V.,
308 Wilson Bldg., Corpus Christi.
Bosquez, Roberto,
4408 Prescott, Corpus Christi.
Bounds, Lloyd D.,
15 Medical Center, Corpus Christi.
Brown, Walter C.,
2550 Morgan, Corpus Christi.
Bryson, J. Gordon, Jr.,
1209 Santa Fe, Corpus Christi.
Burns, Herbert V.,
2430 Morgan, Corpus Christi.
Cameron, Ralph H., Jr.,
2433 Morgan, Corpus Christi.
Carlson, O. Sherman,
1755 Santa Fe, Corpus Christi.
Carr, Robert H., 916 Ayers St., Corpus Christi.
Chriss, John W.,
2436 Morgan St., Corpus Christi.
Clark, Charles S.,
3154 Reid Dr., Corpus Christi.
Clark, Dan H., 1001 Louisiana, Corpus Christi.
Cline, William B., Jr.,
2471 Morgan, Corpus Christi.
Cluff, Frederick M., 401 Cole, Corpus Christi.
Coleman, Sherman T.,
1001 Louisiana, Corpus Christi.
Colyer, George E.,
1001 Louisiana, Corpus Christi.
Concklin, Charles L., Box 3666, Corpus Christi.
Conolly, Sidney M.,
1300 3rd St., Corpus Christi.
Craig, Charles R.,
735 Oak Park, Corpus Christi.
Crain, Carroll F., (Hon.),
1214 Santa Fe, Corpus Christi.
Cullen, Billy B.,
2 Parkdale Plaza, Corpus Christi.
Danford, Edwin A., 712 Booty, Corpus Christi.
Davis, Walter T.,
517 Clifford St., Corpus Christi.
Dixon, Chalmer D.,
P. O. Box 109, Corpus Christi.
Dolch, Stanley F. N., Jr.,
4206 Ayers St., Corpus Christi.
Donada, Maria N.,
2305 Morgan, Corpus Christi.
Draper, Leonidas M.,
207 Wilson Bldg., Corpus Christi.
Eberle, Howard J.,
1201 3rd St., Corpus Christi.
Eckhardt, Kleberg,
3832 Denver, Corpus Christi.
Edwards, Thos. W.,
244 Oleander St., Corpus Christi.
Etheridge, John M.,
1611 Fifth St., Corpus Christi.
Ewing, Dwight S.,
3154 Reid Drive, Corpus Christi.
Feerer, Donald J.,
2 Parkdale Plaza, Corpus Christi.
Fitzgerald, James L.,
3154 Reid Dr., Corpus Christi.
Fitzgerald, Thomas F.,
1001 Louisiana, Corpus Christi.
Flood, George W.,
527 Gordon St., Corpus Christi.
Fordtran, Robert L., (Mil.),
4338 Yucca St., Corpus Christi.
Fosberg, Walter E., (Int.),
Memorial Hospital, Corpus Christi.
Frank, Thelma E.,
1314 16th St., Corpus Christi.
Franks, Edwin R., (Int.),
Memorial Hospital, Corpus Christi.
Fraser, David B.,
1611 5th St., Corpus Christi.
Frich, Michael G.,
525 Everhart Rd., Corpus Christi.
Friedman, Bernard B.,
916 Jones Bldg., Corpus Christi.
Furman, McIver, Furman Bldg., Corpus Christi.
Gabbard, James G.,
1001 Louisiana, Corpus Christi.
Garcia, Clotilde P.,
3838 Baldwin St., Corpus Christi.
Garcia, Hector P.,
3024 Morgan, Corpus Christi.
Garcia, Jose A.,
1101 19th St., Corpus Christi.
Gardner, Joseph E.,
1214 Santa Fe, Corpus Christi.
Garrett, Leslie M.,
2481 Med. Center, Corpus Christi.
Garza, Harold Ross,
1800 S. Staples Bldg., Corpus Christi.
Gentry, William H., (Hon.),
1726 Second St., Corpus Christi.
Ghormley, William C.,
615 Oliver Courts, Corpus Christi.
Giles, E. Jackson,
105 Ocean Way, Corpus Christi.
Gill, E. King, 2414 Morgan, Corpus Christi.
Glanz, Sanford, 2435 Morgan, Corpus Christi.
Goodman, Paul H.,
1214 Santa Fe, Corpus Christi.
Gordon, Robert L., (Int.),
Memorial Hospital, Corpus Christi.
Gray, Paul M., 2420 Morgan, Corpus Christi.
Griffin, Harold E.,
308 Med. Arts Bldg., Corpus Christi.
Groner, Edwin Ben,
533 Gordon, Corpus Christi.
Grossman, Bernard B.,
1001 Louisiana, Corpus Christi.
Grossman, David N.,
Commerce Bldg., Corpus Christi.
Grossman, Maurice S.,
2425 Morgan Ave., Corpus Christi.
Grossman, Saul, 2530 Morgan, Corpus Christi.
Guttman, L. Paul,
1626 10th St., Corpus Christi.
Guttman, Paul B.,
1626 10th St., Corpus Christi.
Gwin, H. Shannon B.,
407 Med. Arts Bldg., Corpus Christi.
Hamilton, Doyne B., Robstown.
Hand, Henry H., (Int.),
Memorial Hospital, Corpus Christi.
Hartwick, Fred W.,
1222 S. Staples, Corpus Christi.
Haynes, Henry M., Jr.,
2418 Morgan, Corpus Christi.
Heaney, H. Gordon,
1300 3rd St., Corpus Christi.
Heaney, Kathryn,
2709 Swantner, Corpus Christi.
Heymann, Hans E.,
2709 Swantner Dr., Corpus Christi.
Hoffpauir, Alvin C.,
Med. Prof. Bldg., Corpus Christi.
Hollingsworth, James S., (Int.),
Memorial Hospital, Corpus Christi.
Holt, Everett L.,
3840 Lexington, Corpus Christi.
House, Rex C., 2455 Morgan, Corpus Christi.
Howze, Jo W., (Mil.), Robstown.
Hubler, Winthrope R.,
1510 S. Brownlee, Corpus Christi.
Hudson, George C., Box 3606, Corpus Christi.
Hudson, Richard L.,
531 Gordon, Corpus Christi.
Hyder, Prentiss L.,
521 Gordon, Corpus Christi.
Janssen, L. W. O., Jr.,
2444 Morgan, Corpus Christi.
Jasperson, Clarence P.,
1326 Santa Fe, Corpus Christi.
Jimenez, Prospero, Jr.,
2554 Morgan, Corpus Christi.
Johnson, Robert W.,
1209 Santa Fe, Corpus Christi.
Jones, John M., 2514 Morgan, Corpus Christi.
Juarez-Reyna, Guillermo,
2731 Morgan St., Corpus Christi.
Kendrick, Michael C.,
2437 Morgan, Corpus Christi.
Kennedy, Hugh A.,
3154 Reid Drive, Corpus Christi.
Kiel, Alois F., 3154 Reid Dr., Corpus Christi.
Klotz, Joseph G.,
2416 Morgan, Corpus Christi.
Knapp, Roger S.,
621 Ohio St., Corpus Christi.
Koepsel, Orlando S.,
2454 Morgan Ave., Corpus Christi.
Kohlhaas, Bernice K.,
417 Miramar Place, Corpus Christi.
Kohlhaas, John K.,
417 Miramar Place, Corpus Christi.
Kurzner, Meyer, 712 Booty, Corpus Christi.
Landesman, Joseph D.,
1234 3rd St., Corpus Christi.
Lane, Alfred L.,
1001 Louisiana, Corpus Christi.
Lang, Rudolph R.,
1001 Louisiana, Corpus Christi.
Lemke, Walter M.,
2522 Morgan, Corpus Christi.
Lingenfelder, John,
3154 Reid Drive, Corpus Christi.
Little, C. R., 1001 Louisiana, Corpus Christi.
Maloney, Vance J., Jr.,
2422 Morgan St., Corpus Christi.
Mann, Nathan,
1806 S. Alameda, Corpus Christi.
Marler, Otis E.,
3216 Reid Drive, Corpus Christi.
Martin, Sterling B.,
1713 S. Brownlee, Corpus Christi.
McCaskey, Gregory M.,
1001 Louisiana, Corpus Christi.
McCaskey, Mary M., (Inac.),
1001 Louisiana, Corpus Christi.
McCutchon, F. James,
3154 Reid Drive, Corpus Christi.
McKemie, Jack F., 527 Gordon, Corpus Christi.
McKenzie, Charles E.,
P. O. Box 3151, Corpus Christi.
Meaney, James J.,
3154 Reid Drive, Corpus Christi.
Mella, Charles A.,
3216 Reid Dr., Corpus Christi.
Metzger, William R.,
2840 S. Alameda, Corpus Christi.
Moller, G. Turner,
2532 Morgan, Corpus Christi.
Moody, Foy H.,
1611 Fifth St., Corpus Christi.
Moon, Orville B., Jr.,
1800 S. Staples Bldg., Corpus Christi.
Moran, J. Patrick,
3154 Reid Dr., Corpus Christi.
Morgan, Chas. G., Jr., (Hon.),
156 Santa Barbara, Corpus Christi.
Morphew, Raymond L.,
1001 Louisiana, Corpus Christi.
Morris, Charles C., Box 6267, Corpus Christi.
Morris, William E.,
1546 S. Brownlee, Corpus Christi.
Nast, Maurice D., 1126 3rd St., Corpus Christi.
O'Brien, Thomas P.,
1001 Louisiana, Corpus Christi.
Oshman, Joseph, 1010 Ohio, Corpus Christi.
Padilla, Arthur,
415 Commerce Bldg., Corpus Christi.
Pasternack, Joseph G.,
1001 Louisiana St., Corpus Christi.
Perkins, Maury J.,
2415 Morgan Ave., Corpus Christi.
Phelps, Travis B.,
1546 S. Brownlee, Corpus Christi.
Pilcher, John F.,
P. O. Box 5416, Corpus Christi.
Posner, Sidney, Robstown.
Powell, Sam M.,
3154 Reid Dr., Corpus Christi.
Priday, Cedric, 934 Ayers, Corpus Christi.
Pruessner, Harold T.,
3840 Lexington Blvd., Corpus Christi.
Rankin, Jean M., (Int.),
Memorial Hospital, Corpus Christi.
Reeves, Gerald A.,
1546 S. Brownlee, Corpus Christi.
Riley, James R., 2400 Morgan, Corpus Christi.
Riley, Winston E.,
2481 Morgan Ave., Corpus Christi.
Rinehart, Archie B., (Inac.),
913 S. Port, Corpus Christi.
Rogers, Fredrick F.,
1001 Louisiana, Corpus Christi.
Roosth, Hyman P.,
2421 Morgan, Corpus Christi.
Rosenheim, Philipp,
2472 Morgan, Corpus Christi.
Russo, G. Martin,
2512 Med. Center, Corpus Christi.
Schulze, John Paul,
1713 S. Brownlee, Corpus Christi.
Schuster, George III,
2427 Morgan, Corpus Christi.
Segrest, John B.,
3218 Olson Drive, Corpus Christi.
Sharp, James C., 2456 Morgan, Corpus Christi.
Shields, William A., Jr.,
2709 Swantner Dr., Corpus Christi.
Sierra, Ernesto, 2735 Morgan, Corpus Christi.
Sigler, Robert J.,
P. O. Box 5212, Corpus Christi.
Sisson, James H., Box 5416, Corpus Christi.
Slabaugh, Carlyle B.,
3154 Reid Drive, Corpus Christi.
Sloan, John J.,
1201 Third St., Corpus Christi.
Sloan, Joseph M.,
P. O. Box 6038, Corpus Christi.
Smith, Youel C., Jr.,
1001 Louisiana, Corpus Christi.
Smith, Youel C., Sr., Box 3606, Corpus Christi.
Sory, Crysup,
1546 S. Brownlee, Corpus Christi.
Spann, R. Gayle,
1546 Brownlee, Corpus Christi.
St. John, Ralph V.,
1001 Louisiana, Corpus Christi.
Stephen, J. J., P. O. Box 231, Robstown.

Stewart, Chas. D.,
1001 Louisiana, Corpus Christi.
Stone, Belo, Robstown.
Stone, Jess C., (Int.),
3222 Huisache, Corpus Christi.
Stone, Lawrence A., Box 308, Robstown.
Stroud, S. K.,
3154 Reid Dr., Corpus Christi.
Swearingen, Robert G.,
1002 Ohio St., Corpus Christi.
Tabler, J. Walton,
1002 Ohio St., Corpus Christi.
Thomas, James H.,
1546 S. Brownlee, Corpus Christi.
Thomas, John R., (Inac.),
702 Morgan, Corpus Christi.
Thomas, Rex E.,
1546 S. Brownlee, Corpus Christi.
Thomason, Elizabeth C.,
2467 Morgan, Corpus Christi.
Thomason, Robert H.,
2467 Morgan St., Corpus Christi.
Triplett, William C.,
1800 S. Staples Bldg., Corpus Christi.
Tyree, James I.,
1209 Santa Fe, Corpus Christi.
Upshaw, Bette Y., (Inac.),
430 Catalina, Corpus Christi.
Upshaw, Jackson E.,
1017 Santa Fe., Corpus Christi.
Veatch, Carolyn K.,
2466 Morgan, Corpus Christi.
Veatch, Donald Hugh,
2466 Morgan Ave., Corpus Christi.
Ware, Stephen H., Jr.,
3154 Reid Dr., Corpus Christi.
Wasson, Robert F.,
1238 Brentwood, Corpus Christi.
Wetegrove, John F.,
2514 Morgan St., Corpus Christi.
White, Hosea A., (Inac.),
412 King St., Corpus Christi.
Wilkens, Robert,
2473 Morgan, Corpus Christi.
Williams, Edmund P., III,
3637 S. Staples St., Corpus Christi.
Williams, Stephen A.,
1201 3rd St., Corpus Christi.
Williford, E. Allan,
1546 S. Brownlee, Corpus Christi.
Wood, Robert C.,
1401 Santa Fe, Corpus Christi.
Woods, Haddon B.,
1335 Third St., Corpus Christi.
Woods, Helen H.,
527 Gordon St., Corpus Christi.
Woods, Richard R.,
2526 Med. Center, Corpus Christi.
Wright, Arthur G., Jr.,
P. O. Box 3606, Corpus Christi.
Wright, L. David,
1546 S. Brownlee, Corpus Christi.
Yates, June, Med. Prof. Bldg., Corpus Christi.
Yeager, Charles P., (Hon.),
408 Wilson Bldg., Corpus Christi.
Yeager, Franklin W.,
408 Wilson Bldg., Corpus Christi.
York, Thomas L., 4206 Ayers, Corpus Christi.
Zinke, Erhardt,
3218 S. Port Ave., Corpus Christi.

SAN PATRICIO-ARANSAS-REFUGIO

Baen, Daniel R., Mathis.
Connolly, John V., Box 787, Ingleside.
Curlee, Curtis L., Sinton.
Deitch, Ernest R., 501 W. Market, Sinton.
Elliott, Boyce,
424 S. Commercial, Aransas Pass.
Ewing, F. Stanley, Sinton.
Farrow, Irving J., 1403 E. Hiller, Victoria.
Finn, John H., Refugio.
Gilmore, Robert M., Portland.
Guilbeau, Joseph A., Jr.,
1403 E. Hiller St., Victoria.
Guynes, William A., Mathis.
Heard, Richard H., Zarsky Bldg., Refugio.
Hudgins, Philip T., Odem.
Jenkins, Young S., 701 Fetick St., Taft.
Koontz, Arch C., Woodsboro.
McConchie, Richard D.,
146 W. Goodnight, Aransas Pass.
Meitzen, Travis C., Refugio.
Miller, Harry A., Refugio.
Pierce, James L., Jr., (Hon.), Box 271, Sinton.
Rittiman, Melross C.,
Ewing Mem. Hosp., Sinton.
Selby, Claude A., P. O. Box 876, Sinton.
Simpson, Charles H.,
501 W. Market St., Sinton.
Stark, Thomas L., 509 W. Market, Sinton.
Tasch, A. F., 717 Field St., Taft.
Tunnell, John W., Taft.
Tunnell, Rose N., Taft.
Voss, Alpheus H., Odem.
Wood, Lloyd G., Rockport.
Zarsky, Emil P., Refugio.

WEBB-ZAPATA-JIM HOGG

Baker, Gordon P., 1607 McClelland, Laredo.
Baker, Julia M.,
Campos Eliseos 81, Mexico City 5, D.F.
Canseco, Francisco M.,
Sames Moore Bldg., Laredo.
Chapa-Badillo, Jesus,
216 Matamoros St., Laredo.
Cigarroa, Joaquin G., Jr.,
708 Matamoros, Laredo.
Cigarroa, Joaquin G., 708 Matamoros, Laredo.
Cigarroa, Leonides G., 806 Matamoros, Laredo.
Cigarroa, Margaret G.,
Sames Moore Bldg., Laredo.
Crawford, James L., (Hon.),
1701 Rosario St., Laredo.
De La Garza, Raul, Sames Moore Bldg., Laredo.
Elizondo, Oscar L., Mercy Hospital, Laredo.
Garcia, George V.,
1915 Convent Ave., Laredo.
Gomez-Rejon, Julio C.,
601 Farragut St., Laredo.
Gonzalez, Blanca C., 1018 Cortex, Laredo.
Gonzalez, Francisco J., Mercy Hosp., Laredo.
Graham, Stephen H., Jr.,
M & S Clinic, Laredo.
Guerra, M. Barrera, Hebbronville.
King, Albert C., 801 Matamoros, Laredo.
Longoria, Enrique M.,
Sames Moore Bldg., Laredo.
Lowry, John T.,
1117 Corpus Christi, Laredo.
Lowry, Ruby S., Sames Moore Bldg., Laredo.
Malakoff, Morris E., 806 Matamoros, Laredo.
Mata, Juan Jose, Valls Clinic, Laredo.
Montalvo, Lauro, 1605 Washington St., Laredo.
Musacchio, Frederick A.,
1011 Hidalgo St., Laredo.
Penny, George E., 801 Matamoros, Laredo.
Perez-Gil, Gerardo, 806 Matamoros St., Laredo.
Powell, William R., 801 Matamoros, Laredo.
Puig, Valentine L., Jr.,
801 Matamoros, Laredo.
Reitman, James S., 2101 Musser St., Laredo.
Rottenstein, Max, Sames Moore Bldg., Laredo.
Trevino, Enrique G., 812 Matamoros, Laredo.
Valls, Miguel, 1004 San Bernardo, Laredo.

SEVENTH DISTRICT

Dr. David Wade, Austin, Councilor

BASTROP-LEE

Burns, Charles M., Box 478, Giddings.
Burns, Robert B., Giddings.
Goddard, Chauncey G., Bastrop.
Hardt, Sterling M., 1109 Church St., Bastrop.
Hazzard, Alford R.,
513 E. Austin St., Giddings.
Koester, Herman L., Box 89, Smithville.
Loveless, Robert W., Box N, Bastrop.
Mantzel, Sherwood W., Box 187, Giddings.
Morris, Roy H., Jr., Fleming Hosp., Elgin.
Reedy, Thomas M., Elgin.
Stephens, J. D., Box 817, Smithville.
Thomas, James W., P. O. Box 747, Smithville.
Weishuhn, Francis J., 302 Olive St., Smithville.

CALDWELL

Fielder, Darwin L., Lockhart.
Luckett, Francis C., Box 13, Fentress.
Nichols, H. Clay, Drawer 111, Luling.
O'Banion, J. Turner,
880 S. Magnolia Ave., Luling.
Playfair, James H., 516 E. Pierce, Luling.
Robertson, Wm. G., Jr., 508 E. Pierce, Luling.
Ross, Abner A., 214 W. San Antonio, Lockhart.
Wales, Philip A.,
214 W. San Antonio, Lockhart.

HAYS-BLANCO

Bell, Charles D.,
422 W. Hutchison, San Marcos.
Bullard, Ray E., Jr., Box 346, Blanco.
Elliott, Benge, 222 W. Hopkins, San Marcos.
Gastring, Joseph B.,
Johnson City Hosp., Johnson City.
Heatly, Maurice D., Box 25, San Marcos.
McCormick, T. Charles, Jr., Buda.
Moore, William L., Jr.,
404 N. Guadalupe, San Marcos.
Primer, B. M., Jr.,
404 N. Guadalupe, San Marcos.
Scheib, Charles W., P. O. Box 11, San Marcos.
Sowell, Rugel F.,
302 W. San Antonio, San Marcos.
Sowell, Rugel F., Jr., Box 76, San Marcos.
Tallant, Arthur N.,
422 W. Hutchison, San Marcos.
White, David L., San Marcos.
Williams, Milton C., (Hon.), San Marcos.

LAMPASAS-BURNET-LLANO

Allen, George S., 410 S. Water St., Burnet.
Brook, Winston M., Box 786, Lampasas.
Carroll, Donald W., 209 East 3rd St., Lampasas.
Dansby, Garland L., Llano.
Gardner, Ralph E., Box 218, Bertram.
Gray, George L., (Hon.), Llano.
Hoerster, Dan J., 110 Main St., Llano.
Hoerster, Henry J., 110 Main St., Llano.
Hotchkiss, Oscar T., 410 S. Water St., Burnet.
McMillin, D. Rush, Lampasas.
Newman, Peggy Jo, Box 128, Burnet.
Ozier, Billy B., 410 S. Water St., Burnet.
Patteson, Morris K., Lampasas.
Shepperd, Joe A., Box 128, Burnet.
Shepperd, Ray L., Box 130, Burnet.
Shepperd, W. Ivan, Marble Falls.
Vaughan, Thomas D., (Inac.), Bertram.
Wood, Alonzo C., Jr., Marble Falls.

TRAVIS

Alexander, Richard J., 720 W. 34th St., Austin.
Allison, Bruce, (Inac.),
5121 Pershing Ave., Fort Worth.
Ambler, Carl D., Student Health Cen., Austin.
Archer, John Dale,
Dept. Health, Education & Welfare,
Washington, D.C.
Archer, Thos. J., Jr.,
2415 Exposition Blvd., Austin.
Auler, Hugo, (Inac.), 4701 Crestway, Austin.
Bachtel, May B., Student Health Cen., Austin.
Baggett, Durward A., (Int.),
Brackenridge Hosp., Austin.
Baggett, Seldon O., 7 Med. Arts Square, Austin.
Bailey, Charles W., 2729 Exposition, Austin.
Bailey, Joe W., 502 W. 15th St., Austin.
Bailey, Walton D., (Int.),
Brackenridge Hosp., Austin.
Bain, Ruth M., 504 W. 17th St., Austin.
Barker, Paul W., 1103 Nueces, Austin.
Barkley, Douglas F., 1702 N. Congress, Austin.
Barnett, Thomas H.,
5314-A Cameron Rd., Austin.
Bates, B. Clary, 1108 Nueces, Austin.
Bethea, James A., (Inac.),
116 Tower Drive, San Antonio.
Blackstock, Mathis W., 2607 La Ronde, Austin.
Blewett, Emerson K., 13 Med. Arts Sq., Austin.
Bohls, Sidney W., 803 E. 32nd St., Austin.
Boston, John A., Jr., 412 W. 17th St., Austin.
Brady, J. J., 1110 Nueces St., Austin.
Brandt, Otto, Jr., 1506 Guadalupe, Austin.
Bratton, Robert E., 1606 Poquonack, Austin.
Brown, Hugh N., (Int.),
State Hosp., Box 96, Austin.
Brown, M. I., Cap. Natl. Bank Bldg., Austin.
Brownlee, Charles H., (Inac.),
1901 W. 35th St., Austin.
Brumage, Wm. S.,
State Health Dept., Austin.
Carter, Rexford G., 1709 San Antonio, Austin.
Chauvin, E. V., 4910 Interregional, Austin.
Clark, George E., Jr., 1 Med. Arts Sq., Austin
Cleveland, G. W., 1209 Parkway, Austin.
Cloud, Ralph E., (Inac.),
48 Summit View, Austin.
Cohen, Ben, 1309 Nueces, Austin.
Coleman, James M., (dead), Austin.
Colwell, Leslie C., 3000 Red River, Austin.
Conner, Beadie E., Box 1004, Austin.
Cooper, R. Allwyn, 924 East 32nd St., Austin.
Covert, Frank M., III, 2903 Red River, Austin.
Crockett, John A., Univ. Health Center, Austin.
Cromer, Horace E.,
605 Cap. Natl. Bank Bldg., Austin.
Crowell, Caroline, 2311 Longview, Austin.
Darnall, Charles M.,
605 Cap. Natl. Bank Bldg., Austin.
Darnall, Joseph R., (Inac.),
3209 Duval St., Austin.
Davidson, Harry T., 1403 Rio Grande, Austin.
Davidson, Morris, (Inac.),
3203 Funsten, Austin.
de Chenar, Coloman,
Austin State Hosp., Austin.
DeGinder, William L.,
2410 Rio Grande, Austin.
Dildy, Charles B., 504 W. 17th St., Austin.
Doles, Emmett A.,
704 Cap. Natl Bank Bldg., Austin.
Douglas, John E., 1403 San Antonio, Austin.
Dryden, Sam H., 1302 Sabine, Austin.
DuBilier, Ben, 528 Capital Natl., Austin.

Dunkelberg, Walter R., (Int.),
Austin State Hosp., Austin.
Dunlop, Josephine N., (Inac.),
108 W. 15th St., Austin.
Eckhardt, James W., 512 Capital Natl., Austin.
Edens, Lee E.,
522 Cap. Natl. Bank Bldg., Austin.
Ellzey, Robert F., 2410 Rio Grande, Austin.
Eppright, Ben R., (Inac.),
2500 Kenmore Ct., Austin.
Esquivel, Sandi, 1306 Rio Grande, Austin.
Exline, Albert L., 505 W. 15th St., Austin.
Farris, Robert G.,
Cap. Natl. Bank Bldg., Austin.
Fatter, Mervin E., 500 W. 15th St., Austin.
Faubion, Darrell B., 2901 Red River, Austin.
Ferrin, Charles E., 1116 Lamar Plaza, Austin.
Forbes, M. Allen, Jr., 607 W. 14th St., Austin.
Fox, Kermit W., 1010 W. 19th St., Austin.
Garcia, Albert G., 2091½ E. 6th St., Austin.
Garcia, John A., 1508 Sabine, Austin.
Gauntt, W. Charles, 811 Nueces, Austin.
Gentry, M. Elizabeth, 2209 Quarry Rd., Austin.
Gilbert, Enid F., 1310 Westmoor Dr., Austin.
Gilbert, Joe T., 918 E. 32nd St., Austin.
Glynn, James D., 707 W. 19th St., Austin.
Goddard, Walter C.,
508 Littlefield Bldg., Austin.
Goehrs, Homer R., 509 W. 26th St., Austin.
Graham, James M., 1504 Guadalupe, Austin.
Grant, Earl L., 4910 Interregional, Austin.
Gregg, F. Banner, 1302 Sabine St., Austin.
Griffin, Lawrence L., 1010 W. 19th St., Austin.
Haan, George W., 3001 Perry Lane, Austin.
Hahn, William B., 1506 Guadalupe, Austin.
Halden, William J., 1424 Berkshire, Austin.
Hall, William Larkin,
4808 Lansing Dr., Austin.
Hamer, James G., 4015 Guadalupe, Austin.
Hanna, Ralph, 108 W. 30th St., Austin.
Hanna, Roger J.,
2698 Heather Dr., East Lansing, Mich.
Hardwicke, Charles P., 918 E. 32nd, Austin.
Harris, Forrest D., (Int.), Box 96, Austin.
Harris, Woodson W.,
126 B East Oltorf, Austin.
Haslund, Thomas M.,
1302 Ridgehaven, Austin.
Hayes, Sigman W., 4804 Grover Ave., Austin.
Heitzman, Celine I. C.,
2600 E. 19th St., Austin.
Helm, Fred P., Tex. State Dept. Health, Austin.
Henry, Harvey B., 1205 Nueces, Austin.
Herrod, James H., 13 Medical Arts Sq., Austin.
Hilgartner, Henry L., Jr.,
202 W. 13th St., Austin.
Hoerster, Samuel A., Jr., Box 96, Austin.
Holland, Lang F., 505 W. 13th St., Austin.
Holtz, Harvey E., 17 Medical Arts Sq., Austin.
Hood, R. Maurice, 10A Med. Arts Sq., Austin.
Hunter, Richard O.,
1512½ S. Congress, Austin.
Jacobs, Maurice, 706 W. 19th St., Austin.
Johnson, Billy Frank, 7 Med. Arts Sq., Austin.
Johnson, David O., 2901 Red River, Austin.
Johnson, J. Edward,
401 Scarbrough Bldg., Austin.
Jordan, Robbie C., 5811 Burnet Rd., Austin.
Kelly, Alfred J., 509 W. 18th St., Austin.
Kelton, William W., Jr.,
108 W. 30th St., Austin.
Key, Sam N., Jr., 120 W. 7th St., Austin.
King, William C., 607 W. 14th St., Austin.
Klint, Hugo A., 1805 Nueces St., Austin.
Klotz, H. L., U. of Tex. Health Cen., Austin.
Kreisle, James E., Box 5158, Austin.
Kreisle, Matthew F., Box 5158, Austin.
Kreisle, Matthew F., Jr., Box 5158, Austin.
Kuenast, Werner E., 5404 W. Hills Dr., Austin.
LaLonde, Albert A.,
Cap. Natl. Bank Bldg., Austin.
Lassiter, James W., 2901 Red River, Austin.
Lawlis, Virgil B., 509 W. 26th St., Austin.
Lawson, Joseph H.,
Brackenridge Hosp., Austin.
Legett, Carey, Jr., 1707 Colorado, Austin.
Legett, Georgia F., 1707 Colorado, Austin.
Legett, Martin P., 3717 East Ave., Austin.
Lippmann, Otto, 9 Med. Arts Square, Austin.
Long, Walter K., 1013 Gaston Ave., Austin.
Lowry, Frederick C.,
6 Med. Arts Square, Austin.
Lucas, Richard A., 502 W. 13th St., Austin.
Martin, Claud A., 1301 Rio Grande, Austin.
Martin, Frances G., (Int.),
2614 W. Jefferson, Dallas.
Martin, Lawrence W., (Mil.),
2614 W. Jefferson, Dallas.
McCauley, Morris D.,
1506A Guadalupe, Austin.
McCorkle, Robert G., Jr.,
405 W. 15th St., Austin.
McCormick, Katharine,
Univ. Health Center, Austin.
McCuistion, C. Hal,
609 Cap. Natl. Bank Bldg., Austin.
McElhenney, Thomas J.,
1402 Nueces St., Austin.
McElhenney, Thos. R.,
1402 Nueces St., Austin.
McIntyre, Francis E., 5718 Burnett Rd., Austin.
McLean, William F., 16 Med. Arts Sq., Austin.
Miller, Clarence R., P. O. Box 96, Austin.
Milligan, Barth, 4616 Red River, Austin.
Moore, Carl F., Jr., 412 E. 5th St., Austin.
Moore, Walter S., 2301 N. Loop, Austin.
Morgan, William P.,
Capital Natl. Bank Bldg., Austin.
Morris, Truman N., 13 Med. Arts Sq., Austin.
Morrison, Robert B.,
801 Cap. Natl. Bank Bldg., Austin.
Murray, R. Vincent, Jr.,
Capital Natl. Bank Bldg., Austin.
Nanney, Audie L.,
406 Cap. Natl. Bank Bldg., Austin.
Neighbors, Allen H., (Inac.),
1801 Lavaca, Austin.
Neighbors, Allan H., Jr.,
926 East 32nd, Austin.
Nelson, F. Murphy, 2 Med. Arts Sq., Austin.
Newman, Henry W., (Inac.),
2710 San Pedro, Austin.
Paggi, Leonard C., 404A W. 15th St., Austin.
Painter, T. S., Jr.,
313 Cap. Natl. Bank Bldg., Austin.
Pape, Robert W., (Int.),
Naval Med. Res. Inst., Bethesda, Md.
Paris, P. J., 811 Littlefield Bldg., Austin.
Paterson, Elizabeth A.,
2614 Delwood Place, Austin.
Patrillo, Albert D.,
510 Cap. Natl. Bank Bldg., Austin.
Paulsen, Paul F., Jr., 2913 Red River, Austin.
Pearce, Francis M., Jr.,
605 Cap. Natl. Bank Bldg., Austin.
Peavy, Charles D., 806 Littlefield Bldg., Austin.
Peavy, J. E., 11908 Oak Trail, Austin.
Pelphrey, Charles F., 8 Med. Arts Sq., Austin.
Phillips, Joseph T., Tribune Bldg., Austin.
Pohl, Donald E., 1 Med. Arts Sq., Austin.
Polsky, Morris, 9 Med. Arts Square, Austin.
Powell, Daniel B., Jr.,
14 Med. Arts Sq., Austin.
Prewett, J. Edwards,
401 Littlefield Bldg., Austin.
Price, Pinckney Clift, 108 W. 30th St., Austin.
Price, Thomas G., 8 Med. Arts Square, Austin.
Primer, Benjamin M., Sr.,
2709 Rio Grande, Austin.
Primer, Charles A., (Int.), Hallettsville.
Queen, Dan M., Brackenridge Hosp., Austin.
Rabb, Virgil S., Star Rt. A, Box 1, Austin.
Rainey, John R., Jr., 8 Med. Arts Sq., Austin.
Ravel, Jerome O.,
6 Med. & Den. Center, Austin.
Reifslager, Walter E., 720 W. 34th St., Austin.
Reinarz, B. H., 511 W. 15th St., Austin.
Roberts, Walter D., 20 Med. Arts Sq., Austin.
Robinson, Harold L., 706 W. 19th St., Austin.
Robison, James T.,
907 Cap. Natl. Bank Bldg., Austin.
Rodriguez, Abe G., (Int.),
1404½ W. 13th St., Austin.
Ross, Raleigh R., 2 Med. Arts Sq., Austin.
Rothen, Robert M., 502 W. 13th St., Austin.
Rude, Joe C., Brackenridge Hosp., Austin.
Ruilmann, Cyril J., 4405 Lamar Blvd., Austin.
Runge, Thomas M.,
907 Cap. Natl. Bank Bldg., Austin.
Salmon, Watt T., (Int.),
Winter V. A. Hosp., Topeka, Kansas.
Sanders, John Nelson,
11 Med. Arts Sq., Austin.
Sayers, Leonard J., 509 W. 26th St., Austin.
Scarborough, Lee F., 720 W. 34th St., Austin.
Schaffner, Leroy, (Int.),
Brackenridge Hosp., Austin.
Schiefelbein, Wm. H., (Int.), Box 96, Austin.
Schiller, Nelson L., 515 W. 15th St., Austin.
Schneider, John P., 1709 San Antonio, Austin.
Schoch, Eugene P., Jr., 112 W. 7th St., Austin.
Scott, H. A., 1302 Sabine, Austin.
Scott, Z. T., (Hon.),
2408 Sweetbrush Dr., Austin.
Sedberry, Margaret M.,
Austin State Hosp., Austin.
Sedberry, Miles E., Jr.,
108 W. 30th St., Austin.
Senter, Jerald R., 4910 Interregional, Austin.
Shaw, R. Preston, (Int.),
708 W. 28th St., Austin.
Simms, Benjamin F., 2905 Red River, Austin.
Smart, V. C., Jr., 1116 S. Lamar, Austin.
Smith, Bobby Joe, (Int.),
4516 Bennet Ave., Austin.
Smith, Howard E., 410 E. 5th St., Austin.
Smith, William S., Jr., (Int.),
Brackenridge Hosp., Austin.
Snider, Robert N., 2410 Rio Grande, Austin.
Sousares, Teddy M.,
7101 Woodrow Ave., Austin.
Stahl, Marion L., 3000 Red River, Austin.
Stenberg, Robert T., Austin State Hosp., Austin.
Stroud, Mary E., (Int.), Box 96, Austin.
Suehs, Oliver W., 14 Med. Arts Sq., Austin.
Swearingen, R. O., 2 Med. Arts Sq., Austin.
Terry, A. A., 918 E. 32nd St., Austin.
Terry, Douglas W.,
907 Cap. Natl. Bank Bldg., Austin.
Thomas, John F., 918 E. 32nd St., Austin.
Thompson, Burch, 2104 Sabine, Austin.
Thompson, Hardy E.,
19th & West Ave., Austin.
Thorne, G. Clifford, 12 Med. Arts Sq., Austin.
Thorne, Lansing S., 12 Med. Arts Sq., Austin.
Thorne, Milner S., 2901 Red River, Austin.
Tipton, George W., 502 W. 15th St., Austin.
Tisdale, Albert A., 20 Med. Arts Sq., Austin.
Tisdale, Marie C., 4205 Caswell Ave., Austin.
Todaro, Samuel P., 706 W. 19th St., Austin.
Turner, Milton, 1015 E. 32nd St., Austin.
Umstattd, Robert G., 2901 Red River, Austin.
Vickers, Albert F., 1201 W. 24th St., Austin.
Wade, David,
510 Cap. Natl. Bank Bldg., Austin.
Walter, Luther P., 3212 Meredith, Austin.
Watkins, W. Pruett, 3313 Hancock Dr., Austin.
Watt, Terrence N.,
304 Cap. Natl. Bank Bldg., Austin.
Watt, Will E.,
508 Cap. Natl. Bank Bldg., Austin.
Weaver, John Dale, 600 W. 11th St., Austin.
Wells, David T., (Int.),
1900 Greenlawn Pky., Austin.
Wharton, Lawrence H.,
602 West 12th St., Austin.
White, B. O., 1707 Nueces St., Austin.
White, Paul L., Health Dept., Texas U., Austin.
White, Wilbur O., 1215B Corona Dr., Austin.
Wilborn, Sam W., 12 Med. Arts Sq., Austin.
Wilks, William N., (Int.),
Box 589, Larned, Kans.
Williams, Harold L., 7 Med. Arts Sq., Austin.
Williams, Harold M., 711 W. 14th St., Austin.
Williams, Harriss,
Capital Natl. Bank Bldg., Austin.
Winkler, John J., 5623 Shoal Creek, Austin.
Womack, David R., 3415 Spanish Oak, Austin.
Womack, Grover K., 1402 Nueces St., Austin.
Woodson, B. Palmer,
Amer. Natl. Bank Arcade, Austin.
Wooten, Greenwood S.,
107 E. 10th St., Austin.
Yeakel, Earl L., Jr., 2901 Red River, Austin.
Yerwood, Connie R., 410 E. Fifth, Austin.
Zedler, Garland G., 603 W. 15th St., Austin.
Zidd, Edward, 403 W. 15th St., Austin.

WILLIAMSON

Alexander, Margaret H., 720 Talbot, Taylor.
Benold, Douglas M.,
703 E. 12th St., Georgetown.
Clearman, Ralph E., P. O. Box 488, Granger.
Cooper, Dewey H., Georgetown.
Daniel, Crawford J., 720 W. 6th St., Taylor.
Gaddy, Howell R., Jr.,
703 E. 12th St., Georgetown.
Gregg, Dick B., Round Rock.
Hermann, Robert C., 813 N. Main, Taylor.
Johns, Jay J., 720 W. 6th St., Taylor.
Kirkpatrick, B. A., Taylor.
Kirkpatrick, Roy H., Taylor.
Lehmberg, Seth W., Taylor.
Leshikar, Marvin J., 720 W. 6th St., Taylor.
Lindley, Gene R.,
308 W. Chandler, Brownwood.
Miller, C. R., Box 26, Leander.
Spires, A. Bryan, Jr., 720 W. Sixth St., Taylor.
Stromberg, Eric W., Taylor.
Swanson, Wayland R., 800 W. 7th St., Taylor.
Wedemeyer, Wm. C., Rt. 3, Georgetown.

EIGHTH DISTRICT

Dr. Carlos E. Fuste, Jr., Alvin, Councilor

BRAZORIA

Brown, G. Bedford, Jr.,
432 Mulberry St., Angleton.
Caldwell, John S., Jr.,
Brazosport Clinic, Freeport.
Carlton, B. Hardy, 326 W. Broad, Freeport.
Carroll, Gay V., Box 26, Lake Jackson.
Coleman, Catherine E.,
Brazoria Co. Health Dept., Angleton.
Cook, T. Edwin, Dow Hospital, Freeport.
Davis, Wallace Rex, Sweeny Clinic, Sweeny.
Fisher, James A., Jr.,
229 Parking Way, Lake Jackson.

Flake, Raymond E., Jr.,
Dow Chemical Co., Freeport.
Fuste, Carlos E., Jr., 907 Gordon, Alvin.
Galloway, William T.,
P. O. Box 1035, Freeport.
Gray, Ralph E., 819 Gulf Blvd., Velasco.
Greenwood, William M., West Columbia.
Hardwick, M. Warren,
839 E. Mulberry, Angleton.
Hayes, Granville J., Alvin.
Heimbigner, Elmer, Box 27, Lake Jackson.
Hester, Fletcher, 409 Main St., Sweeny.
Holt, William C., Box 937, Angleton.
Hooper, Halden W., 508 Main St., Sweeny.
Jamar, Robert S., Jr., 401 Foley, Alvin.
Johnson, Oscar L., Jr.,
120 S. Parking Pl., Lake Jackson.
Kenney, John W., Alvin Mem. Hosp., Alvin.
Kilian, Duane Jack,
208 Oyster Creek, Lake Jackson.
Lee, William M., 815 Hardie St., Alvin.
Lynn, Sherwood C., Brazoria.
Marshall, John E.,
61 Plantation Ct., Lake Jackson.
May, Henry K.,
229 Parking Way, Lake Jackson.
Mays, Jerry D.,
101 Parking Way, Lake Jackson.
McCary, A. O'Brien,
304 W. Broad St., Freeport.
McCary, Rogers M., 304 W. Broad, Freeport.
McDaniel, William, 723 Sealy, Alvin.
McDonald, Richard,
P. O. Box 97, Lake Jackson.
Merz, Herbert E., 815 Hardie, Alvin.
Miller, Robert C., Box J, Lake Jackson.
Montgomery, Joe S., Jr., Box 937, Angleton.
Nelms, William H.,
304 W. Broad St., Freeport.
Nicholson, William D.,
304 W. Broad, Freeport.
Perryman, Gerald F., Dow Hospital, Freeport.
Rau, James R., Box 454, Pearland.
Sears, Warren H., 32 Circle Way, Lake Jackson.
Steele, Wayne K., 933 Mulberry, Angleton.
Stewart, James A.,
229 Parking Way, Lake Jackson.
Turner, Frederick C., Box 127, Lake Jackson.
Venable, John R., Dow Chemical Co., Freeport.

COLORADO-FAYETTE

Boelsche, Leslie D., Box 359, La Grange.
Cummins, James E., Weimar.
DeRuiter, Norman H.,
511 Summit, Schulenburg.
Fuller, Rex G., Jr., Box 188, Weimar.
Guenther, John C.,
P. O. Drawer 269, La Grange.
Ihle, Lyman E., Schulenburg.
Laughlin, John R.,
700 S. McCarty Ave., Eagle Lake.
Laughlin, Jones C.,
700 S. McCarty Ave., Eagle Lake.
Luedemann, William O., (Inac.), Schulenburg.
Makinson, James T.,
851 E. Travis St., La Grange.
Mikesky, Walter E., 715 Lyons St., Schulenburg.
Miller, Arthur C., Carmine.
Paine, Henry C., 851 E. Travis, La Grange.
Peters, Leo J., (Hon.), Schulenburg.
Shult, Clarence I., 418 Wallace, Columbus.
Smith, Herbert T.,
442 E. Guadalupe, La Grange.
Thomas, Raymond R.,
700 S. McCarty Ave., Eagle Lake.
Watzlavick, August J. A.,
P. O. Drawer E, Schulenburg.
Westphal, Corinne, 1227 Bowie, Columbus.
Williams, Edward T., La Grange.
Wilson, James P., 1208 Bowie, Columbus.
Wooten, James H., Jr., Drawer J, Columbus.
Youens, William T., Columbus.
Youens, Willis G., Weimar.
Zatopek, Leland F., 113 Main St., La Grange.

DeWITT

Barth, John H., Yorktown.
Bohman, Alfred J., 106 W. Reuss Blvd., Cuero.
Cross, George W., (Hon.), 4805 Eilers, Austin.
Davis, John C., 615 N. Esplanade, Cuero.
Gohlke, Marvin H., Yorktown.
Hall, Oscar E., Jr., 106 W. Reuss Blvd., Cuero.
High, Harold R., 615 N. Esplanade, Cuero.
Landry, Luchion B., Cuero.
Mgebroff, A. E., Hubbard St., Yoakum.
Milner, Robert M., Yoakum.
Nau, Carl A.,
Univ. of Tex. Med. Br., Galveston.
Nowierski, Leon W., Yorktown.
Prather, Frank A., 106 W. Reuss Blvd., Cuero.
Reuss, Theodore A., 505 N. Esplanade, Cuero.
Richter, Louis B. S., (dead), Yoakum.
Trott, John E., 212 E. Morris St., Yoakum.
Watson, David H., 402 Hubbard, Yoakum.
Westphal, Robert D.,
2002 Holcombe Blvd., Houston.

GALVESTON

Adam, Doris J. D., 3516 Amherst, Dallas.
Adriance, Carroll T., 2402 Sealy, Galveston.
Ainsworth, William H.,
828 Postoffice, Galveston.
Allen, Charles R., 800 Mechanic St., Galveston.
Allensworth, Dan C., III, (Int.),
Univ. of Tex. Med. Br., Galveston.
Allison, James B., (Int.),
112 North Blvd., Galveston.
Anderson, William T.,
421 South Oak, La Marque.
Aves, Fred W., (Inac.),
1501 Ave. J, Galveston.
Baird, Elwood E., John Sealy Hosp., Galveston.
Barber, Ivan J., Jr.,
8117 Highway 6, Hitchcock.
Barker, Julian, (Int.),
P. O. Box 647, Rock Hill, S. C.
Barnes, William B., (Mil.), Gunter AFB, Ala.
Baxter, M. Ruth, 1820 21st St., Galveston.
Baxter, Virgil C., 1820 21st St., Galveston.
Beall, Bobby D., (Int.),
John Sealy Hosp., Galveston.
Beeler, George W.,
9th Ave., N., at 5th, N., Texas City.
Blacklock, David M., 421 S. Oak, La Marque.
Blocker, Truman G., Jr.,
Univ. of Tex. Med. Br., Galveston.
Blocker, Virginia I.,
Univ. of Tex. Med. Br., Galveston.
Boelsche, Arr Nell,
John Sealy Hosp., Galveston.
Box, Quellin T., 915 Strand, Galveston.
Bromberg, Leon, 1501 Broadway, Galveston.
Bruce, E. Ivan, Jr., 1014 Strand, Galveston.
Buchanan, John W., Jr., (Int.),
John Sealy Hosp., Galveston.
Butler, Milton C., Jr., (Int.),
Univ. of Tex. Med. Br., Galveston.
Calma, Victor C.,
Univ. of Tex. Med. Br., Galveston.
Caravageli, M. A.,
506 U. S. Natl. Bank Bldg., Galveston.
Carmignani, Amedeo E.,
819 Avenue J, Galveston.
Casey, Robert E., 822 6th St., N., Texas City.
Charpentier, Leonard A.,
22nd & Sealy, Galveston.
Cherry, James H., 1311 Rosenburg, Galveston.
Childers, John H., P. O. Box 1525, Dallas.
Cochrane, John H., Jr.,
9th Ave. N at 5th, N., Texas City.
Cooke, Willard R., (Emer.),
Univ. of Tex. Med. Br., Galveston.
Cooley, Robert N., John Sealy Hosp., Galveston.
Corssen, Gunter, 928 Strand St., Galveston.
Danforth, Duncan R.,
820 6th St., N., Texas City.
Davis, Harry K.,
Hwy. 3 at Wilkins, League City.
Delany, John J., 22nd & Sealy, Galveston.
deMesquita, Paul B., 2402 Ave. I, Galveston.
Derrick, John R.,
Univ. of Tex. Med. Br., Galveston.
Dolenz, Bernard, (Int.),
1014 Strand, Galveston.
Dudney, Newton E.,
Davis-Dudney Clin., League City.
Duflot, Leo S. M., John Sealy Hosp., Galveston.
Dumas, Lawrence W.,
608 Eighth Ave., N., Texas City.
Dunton, E. Frank, (Int.),
108 Bonita St., Galveston.
Eames, Daniel H., 525 Hwy. 348, La Marque.
Earle, Kenneth M.,
133 San Fernando, Galveston.
Eggers, G. W. N., Jr.,
Univ. of Tex. Med. Br., Galveston.
Eggers, George W. N.,
2201 Avenue D, Galveston.
Emken, Roy Lee, 622 Hwy. 3, La Marque.
Eng, Marlene, (Int.),
Univ. of Tex. Med. Br., Galveston.
Evans, Ernest Burke,
Univ. of Tex. Med. Br., Galveston.
Felton, Harriet M.,
Univ. of Tex. Med. Br., Galveston.
Fisher, William C., (Hon.),
505 S. Prairieville, Athens.
Flanagan, James C.,
Univ. of Tex. Med. Br., Galveston.
Fleming, Ben P., Box 1108, Texas City.
Ford, Charles E., 8117 Hwy. 6, Hitchcock.
Ford, G. David, 525 Hwy. 348, La Marque.
Ford, Hamilton F., John Sealy Hosp., Galveston.
Forman, Sol, 2212 Ave. L, Galveston.
Fowler, M. Lake, Jr.,
4508 Woodrow, Galveston.
Frank, Theo. M.,
American Oil Co., Texas City.
Fuchs, Carl J.,
511 9th Avenue, N., Texas City.
Futch, Edward D.,
611 U. S. Natl. Bank Bldg., Galveston.
Gambrell, William M., Jr.,
831 5th Ave., N., Texas City.
Garbade, Francis A., 22nd & Sealy, Galveston.
Garber, E. Peter, 1501 Broadway, Galveston.
Garza, Marcelo, Jr., (Int.),
Univ. of Tex. Med. Br., Galveston.
George, William K., (Int.),
513 12th St., Galveston.
Gibbs, Reagan H., 1501 Broadway, Galveston.
Gingrich, Wendell D.,
Univ. of Tex. Med. Br., Galveston.
Grant, David Alan, (Int.),
800 Fifth Ave., Fort Worth.
Green, Robert Paul,
823 6th Ave., N., Texas City.
Gregory, Raymond L.,
Med. College, Galveston.
Haggard, Mary Ellen,
John Sealy Hosp., Galveston.
Hander, William W.,
St. Marys Infirmary, Galveston.
Harris, Titus H., John Sealy Hosp., Galveston.
Harrison, A. Wilson,
Univ. of Tex. Med. Br., Galveston.
Hauser, Robert I., (Int.),
142 San Fernando, Galveston.
Headlee, Robert E.,
2018 16th Ave., N., Texas City.
Hejtmancik, Milton R.,
816 Strand St., Galveston.
Henry, Billy W., (Mil.),
USAF Hosp., Sheppard AFB.
Herrmann, George R.,
Univ. of Tex. Med. Br., Galveston.
Holtzclaw, Marjorie L., (Int.),
Univ. of Tex. Med. Br., Galveston.
Hooks, Charles A., 816 Strand, Galveston.
Hopps, Howard C.,
Univ. of Tex. Med. Br., Galveston.
Jackson, Ira J.,
U. S. Natl. Bank Bldg., Galveston.
Jackson, John W., Jr., (Int.),
John Sealy Hosp., Galveston.
Jameson, Grace K.,
112 North Blvd., Galveston.
Jarrell, Norman D.,
822 Sixth St., N., Texas City.
Jarvis, Garth L.,
Univ. of Tex. Med. Br., Galveston.
Jinkins, A. J., 2402 Sealy, Galveston.
Jinkins, Julius L., 22nd & Sealy, Galveston.
Jinkins, Julius L., Jr., 906 22nd St., Galveston.
Jinkins, Wiley J., Jr.,
828 Avenue E, Galveston.
Johnson, James E., Jr., (Mil.),
201D Nolan, Galveston.
Johnson, Jesse B., Jr.,
816 U. S. Natl. Bank Bldg., Galveston.
Johnson, John E., Jr.,
Univ. of Tex. Med. Br., Galveston.
Jones, Edgar F., 2402 Sealy, Galveston.
Joyner, Roy E., 114 21st Ave., N., Texas City.
Kamin, Peter B.,
216 Med. Prof. Bldg., San Antonio.
Kealey, Edward T.,
709 6th St., N., Texas City.
Kirksey, Oscar T., Jr.,
419 U. S. Natl. Bank Bldg., Galveston.
Klein, Elihu I.,
U. S. Natl. Bank Bldg., Galveston.
Klingman, Walter O.,
112 North Blvd., Galveston.
Kolb, Weldon G., 421 S. Oak, La Marque.
Krohn, Walter, St. Marys Hosp., Galveston.
Leavenworth, William M.,
816 U. S. Natl. Bank Bldg., Galveston.
LeBlanc, Alvin L., (Int.),
Univ. of Tex. Med. Br., Galveston.
LeBlanc, Nolan J., Box 398, Cleveland.
Lefeber, Edward J.,
611 U. S. Natl. Bank Bldg., Galveston.
Levin, William C.,
Univ. of Tex. Med. Br., Galveston.
Levine, Harry,
Univ. of Tex. Med. Br., Galveston.
Lewis, Stephen R., John Sealy Hosp., Galveston.
Lionti, Joseph N.,
823 6th Ave., N., Texas City.
Lockhart, Leroy D., (Int.),
107 Dolphin, Galveston.
MacDonald, Etta Mae,
Univ. of Tex. Med. Br., Galveston.
Maddox, Henry E., III,
103 Whiting, Galveston.
Magliolo, Albert M., Dickinson.

Magliolo, Amedeo A., Dickinson.
Magliolo, Andrew J., Dickinson.
Magliolo, Joseph C., Dickinson.
Majors, Irving R.,
9th Ave. & 5th St., N., Texas City.
Mantooth, Gloria E. R., Dickinson.
Marr, William L.,
611 U. S. Natl. Bank Bldg., Galveston.
Martin, Benson L., Jr., (Int.),
Brackenridge Hosp., Austin.
Matlage, William T.,
9th Ave., N., at 5th, N., Texas City.
Mayfield, Demmie G., (Int.),
Univ. of Tex. Med. Br., Galveston.
McDanald, Eugene C., Jr.,
112 North Blvd., Galveston.
McGanity, William J.,
Univ. of Tex. Med. Br., Galveston.
McGivney, John, 2202 Ave. L, Galveston.
McGolrick, Jack B.,
222 Pocahontas Pl., Hampton, Va.
McKenna, Lou Tomlinson,
1102 Avenue E, Galveston.
McLarty, E. Sinks, 1906 21st St., Galveston.
McLarty, E. Sinks, Jr.,
170 San Marino, Galveston.
McReynolds, George S.,
615 North Blvd., Galveston.
Middleton, John W., 900 Strand, Galveston.
Miehl, Morris S., (Int.),
John Sealy Hosp., Galveston.
Moore, Robert M., John Sealy Hosp., Galveston.
Mosely, Mack J., Jr., (dead), Galveston.
Mullins, J. Fred, 927 Strand, Galveston.
Murphy, Joseph G.,
816 U. S. Natl. Bank Bldg., Galveston.
Nau, Carl A., Jr., (Mil.),
Letterman Hosp., San Francisco, Calif.
O'Bryant, Julian W.,
9th Ave., N., at 5th, N., Texas City.
Otto, John L., 112 North Blvd., Galveston.
Parker, James P., 2430 Cedar Dr., La Marque.
Parrish, Beuford R., 1123 Tremont, Galveston.
Patterson, Marcel,
Univ. of Tex. Med. Br., Galveston.
Pearson, Daniel B., Jr., (Int.),
5119 Avenue S, Galveston.
Poth, Edgar J.,
Univ. of Tex. Med. Br., Galveston.
Potter, William B., 1801 Broadway, Galveston.
Powell, L. Charles,
John Sealy Hosp., Galveston.
Prujansky, Nathan, 2201 Ave. D, Galveston.
Quinn, Clarence F., 822 6th St., N., Texas City.
Ramsey, Jack D., (Int.),
John Sealy Hosp., Galveston.
Randall, Edward, Jr.,
205 Natl. Hotel Bldg., Galveston.
Reeves, John P., 820 10th Ave., N., Texas City.
Renshaw, Ralph R., Jr., (Int.),
Hermann Hosp., Houston.
Rigdon, Raymond H.,
Univ. of Tex. Med. Br., Galveston.
Riggs, Stuart, 915 Strand, Galveston.
Ritchie, Earl B.,
Univ. of Tex. Med. Br., Galveston.
Robertson, Gaynelle,
2317 Broadway, Galveston.
Robinson, H. Reid, (Hon.),
810 U. S. Natl. Bank Bldg., Galveston.
Rosenblad, Joanna M.,
830 10th Ave., N., Texas City.
Rosenblad, Lawrence E., Box 171, Texas City.
Ross, Marcus L., 2402 Sealy, Galveston.
Ross, William R.,
City Health Dept., Galveston.
Rowe, Caroline W.,
John Sealy Hosp., Galveston.
Rowe, Edward B., John Sealy Hosp., Galveston.
Ruskin, Arthur,
Univ. of Tex. Med. Br., Galveston.
Sarris, Arthur L., (Int.),
Univ. of Tex. Med. Br., Galveston.
Sarwold, Albert N., 1501 Broadway, Galveston.
Schleuse, Louis W., Jr., (Int.),
Univ. of Tex. Med. Br., Galveston.
Schmidt, Henry A.,
830 10th Ave., N., Texas City.
Schneider, Martin,
Univ. of Tex. Med. Br., Galveston.
Schofield, Norman D.,
John Sealy Hosp., Galveston.
Schwab, Edward H.,
611 U. S. Natl. Bank Bldg., Galveston.
Sharp, William B.,
Univ. of Tex. Med. Br., Galveston.
Singleton, A. O., Jr.,
Univ. of Tex. Med. Br., Galveston.
Slocum, Harvey C., (Mil.),
Walter Reed Hosp., Washington 12, D. C.
Smith, Dan R., 2402 Sealy, Galveston.
Smith, Eugene L., (Inac.), Box 526, Hitchcock.
Smith, James L., (Int.),
106 Tarpon, Galveston.
Smith, Neilson A.,
Davis-Dudney Clin., League City.
Smith, Otto O.,
9th Ave., N., at 5th, N., Texas City.
Snodgrass, Samuel R., 800 Ave. B, Galveston.
Spiller, William F., 22nd & Sealy, Galveston.
Stanton, Elbert M., 501½ 25th St., Galveston.
Stephen, Weldon W., 22nd & Sealy, Galveston.
Stiernberg, Douglas D.,
702 Ninth St., N., Texas City.
Stiernberg, R. Cam,
126 20th Ave., N., Texas City.
Stirling, E. Hopkins,
28 South Shore Dr., Galveston.
Stone, Charles T., Jr.,
U. S. Natl. Bank Bldg., Galveston.
Stone, Charles T., 2201 Ave. D, Galveston.
Stubbs, James B., 1501 Broadway, Galveston.
Sullivan, Robert E., Jr.,
421 South Oak, La Marque.
Sullivan, William W., (Int.),
4510 Caduceus, Galveston.
Sykes, Clarence S., 2201 Ave. D, Galveston.
Thiel, John M.,
Univ. of Tex. Med. Br., Galveston.
Thoma, George W., Jr.,
St. Marys Infirmary, Galveston.
Thompson, Edward R.,
707 U. S. Natl. Bank Bldg., Galveston.
Thornton, William R.,
5 Manor Way, Galveston.
Towler, Martin L.,
John Sealy Hosp., Galveston.
Townsend, Frank M., (Mil.),
Sq. Gr. Hosp., AAF, Washington, D.C.
Tree, Herschel G.,
831 5th Ave., N., Texas City.
Truslow, John B.,
Univ. of Tex. Med. Br., Galveston.
Tucker, John R., (Int.),
John Sealy Hosp., Galveston.
Tucker, William J., (Mil.),
Fairfield AFB, Spokane, Wash.
Twidwell, Leonard,
715 10th Ave., N., Texas City.
Verrett, Richard R.,
66 3rd Ave. Villas, Texas City.
Vogelpohl, Elmer B., Jr., (Int.),
Univ. of Tex. Med. Br., Galveston.
Walker, Joe A.,
Univ. of Tex. Med. Br., Galveston.
Warren, James V.,
Univ. of Tex. Med. Br., Galveston.
Webb, John A.,
Univ. of Tex. Med. Br., Galveston.
Weinert, Herman, Jr.,
710 U. S. Natl. Bank Bldg., Galveston.
Weiss, Victor J., Jr., (Int.),
John Sealy Hosp., Galveston.
Wellman, Garland O.,
6th St. at 9th Ave., Texas City.
Williams, G. Douglas,
P. O. Box 487, La Marque.
Williams, Raymond E., (Int.),
John Sealy Hosp., Galveston.
Williams, Thomas B., (Int.),
John Sealy Hosp., Galveston.
Wilson, Charles J., 3801 Ave. N, Galveston.
Wilson, McClure,
2704 Houston Dr., S., La Marque.
Wilson, William A., Hotel Galvez, Galveston.
Wilson, William P., 1014 Strand, Galveston.
Wolma, Fred J., Jr.,
120 Barracuda Ave., Galveston.
Wright, James C., Jr.,
6713 Fairway Dr., Galveston.
Yen, Moore,
Univ. of Tex. Med. Sch., Galveston.

LAVACA

Boyle, James W., (Inac.),
5015 Highland Ct., Austin.
Gaddis, Herman W.,
302 E. Second, Hallettsville.
Marek, Emil H., Yoakum.
Renger, Harvey, Hallettsville.
Wagner, John D., Shiner Clinic, Shiner.
Wagner, Robert J., Shiner Clinic, Shiner.
Williams, Robert W., Shiner.
Yates, Loren K., Box 434, Hallettsville.

VICTORIA-CALHOUN-GOLIAD

Allen, Richard C., Jr.,
1702 N. Laurent, Victoria.
Avant, W. Harold, (Mil.),
1508 E. Mimosa St., Victoria.
Bade, Craig P., 2006 N. Navarro, Victoria.
Bauer, James E., Box 3344, Victoria.
Bickford, Colon U., 2007 N. Laurent, Victoria.
Bolton, Billy F., 2701 N. Azalea, Victoria.
Borchers, Charles L.,
2006 N. Navarro, Victoria.
Bridges, Doye R., 2701 N. Azalea, Victoria.
Cogswell, Jack W., 2701 N. Azalea, Victoria.
Cohen, Kenneth, (Mil.),
Foster AFB, Victoria.
Coleman, James L., Jr., Box 3346, Victoria.
Coleman, Winton L., 2203 Retama, Victoria.
Constant, George A., 306 N. Moody, Victoria.
Crabtree, Jerry W., 401 E. Murray St., Victoria.
Crenshaw, Earl S., Box 500, Point Comfort.
Dodson, Pattie May,
1801 N. Laurent St., Victoria.
Duckworth, Frederick L.,
2104 Loma Vista, Victoria.
Ehlert, Edward A., Jr.,
121½ S. Main, Victoria.
Gilliam, Rochell B., 1801 N. Laurent, Victoria.
Glover, George E., Jr.,
2008 N. Navarro, Victoria.
Griffin, John W., 117 Ash, Port Lavaca.
Herbert, Joseph W.,
2701 N. Azalea St., Victoria.
Hicks, William M., Jr.,
2701 N. Azalea St., Victoria.
Hilderbrand, Harold E., Box 502, Goliad.
Hopkins, Joseph V., (Hon.),
420 Natl. Bank Bldg., Victoria.
Horine, Maurice S., 117 Ash St., Port Lavaca.
Jones, Lloyd C., 2701 N. Azalea, Victoria.
Kleiman, Harold, 2707 N. Laurent, Victoria.
Lancaster, York, 623 N. Virginia, Port Lavaca.
Lander, Roy S., 1501E N. DeLeon, Victoria.
Lester, Stanley W.,
717 N. Virginia, Port Lavaca.
Logsdon, Francis M.,
P. O. Box 551, Port Lavaca.
Martin, Thomas Lee,
2701 N. Azalea, Victoria.
McCollum, C. J., 402 E. Guadalupe, Victoria.
McGlothlen, George E., Box 3347, Victoria.
Messer, Clarence R., Seadrift.
Mooney, Ern C., 2008 N. Navarro, Victoria.
Norton, John A., (Mil.), Foster AFB, Victoria.
Obert, Paul M.,
Citizens Mem. Hosp., Victoria.
Reed, Roy G.,
Victoria Co. Health Dept., Victoria.
Reiner, Robert N., (Mil.),
Foster AFB, Victoria.
Sale, Walter W., Natl. Bank Bldg., Victoria.
Seger, Eva Y., 108 E. Loma Vista, Victoria.
Seger, Forrest M., 2008 N. Navarro, Victoria.
Shields, Allen H., 1301 N. Bridge, Victoria.
Smith, D. Heaton, 1501A N. DeLeon, Victoria.
Smith, William G.,
206 S. Guadalupe, Port Lavaca.
Southern, Jack, (Mil.),
548 S. 2nd St., Bellaire.
Sterne, Thomas L., 2701 N. Azalea, Victoria.
Stevenson, Rufus A., Jr.,
2005 N. Navarro St., Victoria.
Story, Joseph R., 1501D N. DeLeon, Victoria.
Walker, John H.,
E. I. duPont de Nemours, Victoria.
Ward, Rawley W.,
401 W. Stayton Ave., Victoria.
White, Ben Henson, 1702 N. Laurent, Victoria.
Wilhite, Hilton R., 2601 N. Azalea, Victoria.

WHARTON-JACKSON-MATAGORDA-FORT BEND

Amman, Franz E., 806 3rd, Rosenberg.
Arbuckle, Bertrand D.,
201 E. Jackson, El Campo.
Avalos, Sergio A., 806 Third St., Rosenberg.
Bader, Joseph N., Box 308, Edna.
Barbour, J. Lane, 2417 Avenue G, Bay City.
Barnes, Harry T., Jr., 1015 Ave. G, Bay City.
Bauknight, James M., Ganado.
Beavers, Fred W., 1112 Second St., Rosenberg.
Bishop, Charles L., Box 545, Ganado.
Black, Vernon A., 105 Hawes St., Wharton.
Blasingame, F. J. L., (Hon.),
535 N. Dearborn St., Chicago, Ill.
Boswell, Samuel R., P. O. Box 72, Rosenberg.
Brewer, Earl J., Jr., 210 N. Houston, Wharton.
Brewer, Paul L., Box 308, Bay City.
Cannon, Joseph N., Jr., Box 1467, Bay City.
Caraway, Robert B., 105 Hawes St., Wharton.
Cowart, James E., Box 1467, Bay City.
Doss, George W., Box 846, Edna.
Dye, Fulton E., 2230 Avenue F, Bay City.
Fretz, Howard Z., 105 Hawes St., Wharton.
Hadnott, William H., 2500 Ave. C, Bay City.
Halamicek, John A., Box 167, El Campo.
Halamicek, John F., Box 167, El Campo.
Hoch, Rodney, 600 N. Wells, Edna.
Horton, Granville E.,
218 N. Houston, Wharton.
Jackson, Robert H., Jr., 2230 Ave. F, Bay City.
Johnson, Leonard B., El Campo.
Johnson, R. G., New Gulf.

Kaplan, Hyman J., 1100 Avenue G, Bay City.
Kleinman, Isaac, 3203 Ave. H, Rosenberg.
Knolle, Ben E., 1300 First St., Rosenberg.
Kolle, Fred W., 105 Hawes St., Wharton.
Kuykendall, Harold D.,
P. O. Box 27, Sugar Land.
Leazar, Marshall A., 218 N. Houston, Wharton.
Leslie, Robert E.,
606 W. Jackson St., El Campo.
Little, Raymond D., 105 Hawes St., Wharton.
Mangum, Hugh J., Rt. 1, Box 636, Brazoria.
Matthes, Fred T., 1801 Merlin, Bay City.
Matthes, Homer C., 1100 Ave. G, Bay City.
McGee, Borden M., Box 365, College Station.
McKinney, William C., Box 696, Edna.
McMasters, Stanley R., Box 910, El Campo.
Meadows, Charles T., 105 Hawes St., Wharton.
Nichols, C. V., 202 4th St., Richmond.
Northington, Harold M.,
218 N. Houston, Wharton.
Outlar, L. Bolton, 218 N. Houston, Wharton.
Presley, Walter D., El Campo.
Reeves, Hiram V., (Hon.), El Campo.
Sanford, E. B., Box 1488, Palacios.
Sawyers, James R., Bader-Sawyers Clin., Edna.
Schuhmann, J. Daniel, East Bernard.
Scott, Will E., P. O. Box 72, Rosenberg.
Secor, Harold E., 105 Hawes St., Wharton.
Shoultz, Charles A., Box 993, Bay City.
Simons, Bryan E., Bay City.
Simons, James W., New Gulf.
Stengl, Lorraine I., Box 429, El Campo.
Stepan, John D.,
206 W. Monsarette, El Campo.
Thiltgen, Winston S., El Campo.
Thompson, Stanley E., 202 4th St., Richmond.
Voelker, Chris A., P. O. Box 548, Wharton.
Voulgaris, Dennis M., 105 Hawes St., Wharton.
Wilcox, Leroy A., Palacios.
Witt, Raymond R., 210 N. Houston, Wharton.
Woodson, Clinton E., 105 Hawes St., Wharton.
Yelderman, Gus C., Box 326, Rosenberg.
Yelderman, Joe C., Needville.
Yelderman, Robert L.,
1034 2nd St., Rosenberg.

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Neely, Jubal A., Bellville.
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GRIMES

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500 E. Washington, Navasota.
Coleman, Solon D., Drawer 311, Navasota.
Conkling, William Scott,
500 E. Washington, Navasota.
Galbreath, John C.
503 E. Washington, Navasota.
Hansen, C. Marius, Box 227, Navasota.
Ketchum, Everard T., Navasota.
Parker, Marshall E., (Hon.), Anderson.

HARRIS

Abbott, Jack P., 6516 Bertner Blvd., Houston.
Able, Luke W.,
921 Hermann Prof. Bldg., Houston.
Abolafia, Max, 3016 Canal St., Houston.
Adam, George F., 4510 Caroline St., Houston.
Adamo, Dominick C.,
411 W. 20th St., Houston.
Ainsworth, Joseph T., 6515 Lawndale, Houston.
Alexander, Charles S.,
Hermann Prof. Bldg., Houston.
Alexander, George G.,
1029 E. Thomas, Pasadena.
Alexander, Herbert L., 2918 Caroline, Houston.
Alexander, James K.,
1200 M. D. Anderson Blvd., Houston.
Alford, Bobby R., (Int.),
4123 S½, Galveston.
Alford, Jack A.,
4562 Old Richmond Rd., Bellaire.
Alger, Leon J., Pasadena Prof. Bldg., Pasadena.
Allen, Herbert C., Jr.,
100 Hermann Prof. Bldg., Houston.
Allen, Leonardo, (Inac.),
Medical Arts Bldg., Houston.
Alvord, Ellsworth C., Jr.,
1200 M. D. Anderson Blvd., Houston.
Ameen, Ray C.,
8849 Long Point Rd., Houston.
Anchell, Melvin,
9129 E. Houston Rd., Houston.
Anderson, A. Burton,
1030 Hermann Prof. Bldg., Houston.
Anderson, Thomas A., Jr.,
2708 Weslayan, Houston.
Andres, David, 906 E. Southmore St., Pasadena.
Andrew, John G., 904 E. Southmore, Pasadena.
Andrew, Leora P., 8060 Moline, Houston.
Andrews, John H., 719 E. Texas Ave., Baytown.
Andrews, Tom A., Jr.,
1017 Hermann Prof. Bldg., Houston.
Andronis, Aristos C.,
1105 Med. Towers, Houston.
Antony, O. Owen, 2921 Luell St., Houston.
Applebe, Edward W., (Hon.),
1806 Holman, Houston.
Arango, George A., 1110 Hogan St., Houston.
Archer, Francis C., 2800 Garth Rd., Baytown.
Armbrust, Charles A., Jr.,
4109 Montrose Blvd., Houston.
Armstrong, John T.,
Hermann Prof. Bldg., Houston.
Armstrong, Willie O., Box 2197, Houston.
Arnold, Hiram P.,
Hermann Prof. Bldg., Houston.
Arnold, Hugh F., 6448 Fannin, Houston.
Arnold, Jasper H.,
1611 Med. Towers, Houston.
Arnold, W. Tom, 6448 Fannin, Houston.
Ashkenazy, Moses, 910 Med. Towers, Houston.
Ashmore, Charles M.,
1411 Mem. Prof. Bldg., Houston.
Atkins, Richard D., Jr.,
3229 Wisconsin, Baytown.
Atlas, Joe, 5408 Palms Center, Houston.
Atlas, Leon T., 624 Tatar, Pasadena.
Avery, Eldridge F.,
906 E. Southmore, Pasadena.
Aves, Frederick H.,
1215 Hermann Prof. Bldg., Houston.
Axelrod, Alexander,
1009 Med. Arts Bldg., Houston.
Axelrod, William, 1109 Med. Towers, Houston.
Babcock, Darrow S., 2921 Luell St., Houston.
Bacon, Robert J.,
5101 Lockwood Dr., Houston.
Baechtel, William R., (Int.),
Hermann Hospital, Houston.
Bains, Louis W., 2030 Colquitt, Houston.
Baird, Raleigh W., Jr.,
3701 Montrose Blvd., Houston.
Baird, Val C., 382 Humble Bldg., Houston.
Baker, Lowell B., 1805 Crawford, Houston.
Ballantyne, A. J., 6723 Bertner Dr., Houston.
Bancroft, Chas. E., 4828 Caroline, Houston.
Banfield, Edison H., 2914 Blodgett, Houston.
Banfield, Michael D., 3004 Holman, Houston.
Barfield, Frank T., Jr., (Mil.),
NAAS, Chase Field.
Barkin, Gilbert D.,
3509 Montrose Blvd., Houston.
Barkley, Howard T.,
4414 Montrose Blvd., Houston.
Barkley, Howard T., Jr.,
4414 Montrose Blvd., Houston.
Barnes, J. Peyton, 617 Med. Towers, Houston.
Barnett, Herman A., 5101 Lockwood, Houston.
Barnhart, Joseph M.,
1817 Dryden Rd., Houston.
Barrett, John H.,
1600 Mem. Prof. Bldg., Houston.
Barry, William E., 2708 Weslayan, Houston.
Barton, Harry L.,
2002 Holcombe Blvd., Houston.
Bash, Vincent C., Jr.,
8849 Long Point Rd., Houston.
Batmanis, Elizabeth,
4010 Montrose Blvd., Houston.
Bayles, Spencer, 512 Med. Towers, Houston.
Beall, Arthur C., Jr., (Int.),
3914 Purdue, Houston.
Beard, Earl F., 6655 Travis, Houston.
Behr, Thomas S., 5620 Westheimer, Houston.
Behrens, Charles A.,
5644 Lawndale St., Houston.
Bell, Exter F., Jr., 1721 Med. Towers, Houston.
Bell, Justin E., 2255 W. Holcombe, Houston.
Belleggie, Philip A.,
5019 Westheimer, Houston.
Bennett, Hugh D.,
2002 Holcombe Blvd., Houston.
Bennett, William H., (Hon.), Humble.
Benson, William F.,
906 E. Southmore, Pasadena.
Bergsagel, Daniel E.,
6723 Bertner Dr., Houston.
Bernard, Lynn A.,
Hermann Prof. Bldg., Houston.
Bertinot, Chapman W.,
1100 E. Holcombe Blvd., Houston.
Berumen, Joe F.,
1700 Holcombe Blvd., Houston.
Berumen, Thelma C.,
317 Park Towers Bldg., Houston.
Best, Paul W., (Inac.),
3665 Wickersham, Houston.
Bettis, Moody C., 706 Med. Towers, Houston.
Betts, William A., Jr.,
3102 S. Harvard, Tulsa, Okla.
Bevil, Jack N.,
1528 Mem. Prof. Bldg., Houston.
Beyer, Alvin, Jr.,
1521 Mem. Prof. Bldg., Houston.
Bickel, Laura C., 30 Oakdale, Houston.
Biles, E. Wiley, 6621 Fannin St., Houston.
Billups, J. T., Hermann Prof. Bldg., Houston.
Bing, Arlys W., P. O. Box 40095, Houston 40.
Bing, Lyndon W., Katy.
Birdsell, John E.,
8411 E. Houston Rd., Houston.
Blackburn, Edward A., Jr.,
1600 Mem. Prof. Bldg., Houston.
Blair, Lyman C., 1212 Rothwell, Houston.
Blair, Robert K., 2418 Travis, Houston.
Blattner, Russell J.,
Baylor College of Med., Houston.
Bloom, Fred A., (dead), Houston.
Bloom, Manuel G., 912 Med. Towers, Houston.
Bloxsom, Allan P., 4402 Travis St., Houston.
Blum, Morton A., 6410 Fannin St., Houston.
Blundell, J. Reese,
302 Med. Arts Bldg., Houston.
Boice, Edward H.,
6303 S. Park Blvd., Houston.
Bonham, Russell F., Box 6237, Houston.
Bonin, Wilfred P., 1601 W. Alabama, Houston.
Boone, Carroll L., 906 E. Southmore, Pasadena.
Boone, James B.,
Southmore Hosp.-Clin., Pasadena.
Boren, James S.,
1227 Mem. Prof. Bldg., Houston.
Bourdon, Lynn L., 414 W. 20th St., Houston.
Bowen, Shirley S.,
1408 Hermann Prof. Bldg., Houston.
Bowie, Albert Ely, 3117 Dowling St., Houston.
Bowie, Carl W.,
3701 Montrose Blvd., Houston.
Bowles, John D., 618 Prairie, Houston.
Boyd, Adam N., 2918 Caroline St., Houston.
Boyd, Charles D., 6717 Sherwood, Houston.
Braastad, Frederick W.,
Hermann Prof. Bldg., Houston.
Braden, Albert H., Jr.,
No. 2 Chelsea Pl., Houston.
Braden, David J., No. 2 Chelsea Pl., Houston.
Bradford, F. Keith,
435 Hermann Prof. Bldg., Houston.
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Bragg, William J., Jr.,
4717 Laura Koppe, Houston.
Brandau, George H.,
619 Med. Arts Bldg., Houston.
Brandes, E. B.,
1700 E. Holcombe Blvd., Houston.
Brandon, Sylvan,
703 Med. Arts Bldg., Houston.
Brannon, Jack G., 2715 Fannin St., Houston.
Bratteng, Elizabeth H.,
8916 Spring Branch Dr., Houston.
Brauer, Raymond O., 6615 Travis, Houston.
Braun, Howard E., 5900 Lyons Ave., Houston.
Breckenridge, Carlyle G.,
2014 Crawford, Houston.
Brelsford, Harold J.,
4062 Tartan Lane, Houston.
Brener, Lazard S., 2219 Dorrington, Houston.
Brener, Zidella S., 2219 Dorrington, Houston.
Bressler, J. L.,
1511 Med. Arts Bldg., Houston.
Brewer, Raymond L.,
6800 Long Point Road, Houston.
Brewer, Thomas W.,
910 Hermann Prof. Bldg., Houston.
Bridges, William H., 504 W. Defee, Baytown.
Brinsmade, Alan B., (Int.),
2721 Kipling, Houston.
Brockman, H. Leroy, Jr.,
1012 Med. Towers, Houston.
Brodsky, Alexander E.,
505 Med. Towers, Houston.
Brooks, Benjy F.,
1506 Med. Towers, Houston.
Brown, Jack Ross, 1010 Federal Rd., Houston.
Brown, James A., 5101 Almeda, Houston.
Brown, John H., 1319 S. Richey, Pasadena.
Brown, John R.,
3004 Little York Rd., Houston.
Brown, R. Alex, 7401 Harrisburg, Houston.

Brown, William K.,
Hermann Prof. Bldg., Houston.
Brown, Wilson G.,
165 Hermann Prof. Bldg., Houston.
Broyles, George D., Jr.,
Hermann Prof. Bldg., Houston.
Bruce, George D., 721 E. Texas Ave., Baytown.
Bruhl, Charles E., (Hon.),
1706 North Blvd., Houston.
Bruhl, Charles K.,
345 Hermann Prof. Bldg., Houston.
Bryan, W. G., 1401 Eagle St., Houston.
Bucci, Robert J.,
1823 Mill Creek Dr., Houston.
Bukowski, John,
532 N. Esperson Bldg., Houston.
Bukowski, Lucian M., 1814 Crawford, Houston.
Buldain, Martha, 4510 Caroline St., Houston.
Bullock, Elza V., 906 E. Southmore, Pasadena.
Bunting, John J.,
4705 Montrose Blvd., Houston.
Burch, Neil R.,
1200 M. D. Anderson Blvd., Houston.
Burdeaux, Billie D., Jr.,
815 Med. Arts Bldg., Houston.
Burden, Alfred L., Jr., (Int.),
1203 Ross Sterling, Houston.
Burdick, Jesse G., Ethyl Corporation, Pasadena.
Burg, Abner D., 806 Med. Towers, Houston.
Burge, Curtis H.,
Hermann Prof. Bldg., Houston.
Burke, Thomas W., Esperson Bldg., Houston.
Burkhalter, Archie H.,
906 E. Southmore, Pasadena.
Burnett, Edmund L.,
1109 Hermann Prof. Bldg., Houston.
Burnett, Mathew D., Jr.,
Hermann Prof. Bldg., Houston.
Burr, Harry B.,
636 Hermann Prof. Bldg., Houston.
Burrows, John B.,
Hermann Prof. Bldg., Houston.
Butera, J. M., 1814 Crawford St., Houston.
Butler, Donald B.,
1509 Med. Towers, Houston.
Butler, Max C., 6154 Westheimer, Houston.
Cady, Lee D., V. A. Hospital, Houston.
Cain, Donald N., 904 E. Southmore, Pasadena.
Calhoun, C. Alsworth,
824 Hermann Prof. Bldg., Houston.
Cameron, Bruce M., 5220 Travis St., Houston.
Campbell, G. Maury, (Inac.),
Hermann Prof. Bldg., Houston.
Candis, Dean J.,
4520 Rossmoyne Blvd., Houston.
Cantrell, Wm. Allen,
706 Med. Towers, Houston.
Caplan, Mischa, 2219 Dorrington, Houston.
Caplovitz, Coleman D.,
1120 Med. Towers, Houston.
Caplovitz, Harry, 1120 Med. Towers, Houston.
Cappleman, William P.,
203 Bayshore Dr., Baytown.
Caram, Pedro, 1708 Med. Towers, Houston.
Carlton, Lawrence E.,
4119 Montrose Blvd., Houston.
Carrigan, E. W., Jr., 106 W. Jackson, Pasadena.
Carrington, Dewitte C.,
4010 Montrose Blvd., Houston.
Carrithers, Clem M., 2921 Luell St., Houston.
Carroll, Carl M., Jr., 3921 Lyon Ave., Houston.
Carroll, James A., 1801 Med. Towers, Houston.
Carroll, Roland B.,
1223 Mem. Prof. Bldg., Houston.
Carter, Edgar H., 5102 N. Main St., Houston.
Carter, James D., 2034 Sheridan, Houston.
Carter, Margaret A.,
2531 Dryden Rd., Houston.
Carter, R. Edward, Jr.,
1701 S. Shepherd Dr., Houston.
Caruso, Charles J.,
6303 S. Park Blvd., Houston.
Cato, Dorothy A.,
1400 Hermann Dr., Houston.
Cavazos, Ninfa, 4206 Yoakum Blvd., Houston.
Cecala, Philip J.,
2319 S. Shepherd Dr., Houston.
Chalmers, Presley H., 2310 Gramercy, Houston.
Chamberlin, James Allen,
1501 Hermann Prof. Bldg., Houston.
Chandler, Edwin A., 6611 Travis, Houston.
Chao, Dora Hsi Chih,
Methodist Hospital, Houston.
Chapin, Elisha H., Rice Hotel, Houston.
Chapman, Don W.,
Hermann Prof. Bldg., Houston.
Chau, Paul M. 6723 Bertner Ave., Houston.
Chernosky, Marvin E.,
Hermann Prof. Bldg., Houston.
Chunn, Edward K., 914 Med. Towers, Houston.
Clark, R. Lee, Jr.,
6723 Bertner Blvd., Houston.
Clark, William A.,
3407 Montrose Blvd., Houston.
Clarke, Edward T., 2404 Rice Blvd., Houston.
Clarke, Herndon H.,
2015 Dryden Rd., Houston.
Clarke, Jared E.,
713 Med. Arts Bldg., Houston.
Clarke, Thomas P., 514 Med. Towers, Houston.
Clarkson, Ira S.,
1206 Mem. Prof. Bldg., Houston.
Clause, Harry P., Jr., (Int.),
11407 Landsdowne, Houston.
Clayton, George W.,
1200 M. D. Anderson Blvd., Houston.
Cline, Deane T.,
504 Hermann Prof. Bldg., Houston.
Cloud, Thomas M., 613 Med. Towers, Houston.
Cochran, Chester G., 6655 Travis, Houston.
Cody, Claude C., III,
1616 Mem. Prof. Bldg., Houston.
Cody, James R., 4951 Glenmeadow, Houston.
Cody, Melville L., 4115 Fannin St., Houston.
Coe, Richard O., Jr., (Int.),
5763 W. Airport Blvd., Houston.
Cogan, John E., (Int.),
Hermann Hospital, Houston.
Cogburn, Charles C., 617 Tatar St., Pasadena.
Cohan, Stuart K.,
6417 Richmond Rd., Houston.
Cohen, Irvin M.,
Hermann Prof. Bldg., Houston.
Cohen, Raymond,
1700 E. Holcombe, Houston.
Cole, V. William, (Int.),
1200 M. D. Anderson Blvd., Houston.
Cole, W. Frank,
112 Meyerland Plaza, Houston.
Coleman, Ruth, 1516 Pierce St., Houston.
Collette, Allan,
1006 Med. Arts Bldg., Houston.
Collier, James L.,
8849 Long Point Rd., Houston.
Collins, George P., 618 S. Tatar, Pasadena.
Collins, Lois Cowan,
M. D. Anderson Hosp., Houston.
Collins, Ray G., 718 Med. Arts Bldg., Houston.
Collins, Vincent P.,
1200 M. D. Anderson Blvd., Houston.
Colpitts, R. Vernon,
703 The Med. Towers, Houston.
Compere, Thomas H.,
2631 Fenwood Rd., Houston.
Comstock, Betty S.,
6235 Beechnut St., Houston.
Connor, Edwin E.,
2303 Fenwood, Pasadena.
Connor, W. Harris,
1220 Med. Arts Bldg., Houston.
Conte, Raphael J., 3517 Louisiana, Houston.
Cooley, Denton A.,
Baylor Med. College, Houston.
Coons, Robert E., (Mil.),
4515 Normandy Way, Houston.
Cooper, Jack C., 1109 S. Shepherd, Houston.
Cope, R. Louis, 713 Med. Arts Bldg., Houston.
Corbett, Louis B., 3507 Crawford, Houston.
Cornelius, Eugene A.,
Hermann Hospital, Houston.
Corpening, Thomas N.,
10521 S. Post Oak Rd., Houston.
Cotlar, Nathan, 711 Med. Towers, Houston.
Coulter, William W., Sr.,
1215 Walker Ave., Houston.
Cowart, Edmund M., 10 Chelsea Pl., Houston.
Cowart, Marolyn M.,
8849 Long Point Rd., Houston.
Cox, Dewitt W., Jr.,
1429 Med. Prof. Bldg., Houston.
Cox, James F., Jr., 2701 Berry Rd., Houston.
Cox, John K.,
1508 Hermann Prof. Bldg., Houston.
Cox, Joseph H., 618 Tatar, Pasadena.
Crain, Edward L., Jr.,
Hermann Prof. Bldg., Houston.
Crain, Lovell B.,
Hermann Prof. Bldg., Houston.
Crank, H. Harlan,
805 N. Post Oak Rd., Houston.
Crapitto, Louis A.,
1113 Med. Arts Bldg., Houston.
Crawford, E. Stanley,
1200 M. D. Anderson Blvd., Houston.
Crawford, Elisabeth S.,
Hermann Prof. Bldg., Houston.
Crawley, James W.,
1200 M. D. Anderson Blvd., Houston.
Crigler, Cecil M.,
Hermann Prof. Bldg., Houston.
Crocker, Edward S., 2121 San Jacinto, Houston.
Cromwell, Henry A.,
5000 Montrose Blvd., Houston.
Cronin, Thomas D., 6615 Travis St., Houston.
Crouch, Robert B., 712 Med. Towers, Houston.
Crow, Howard G., 809 Med. Towers, Houston.
Crow, Louise C., 4930 Braes Valley, Houston.
Cruce, William V.,
1407 N. Shepherd, Houston.
Cull, Herbert G.,
8518 Humble Rd., Houston.
Cullen, Aubrey P., Jr.,
7410 Long Point Rd., Houston.
Cullick, Louis, 737 S. 75th St., Houston.
Culotta, Ralph J., 624 Tatar St., Pasadena.
Cummings, Hatch W.,
Hermann Prof. Bldg., Houston.
Cunningham, G. Norman,
Esperson Bldg., Houston.
Cunningham, James E.,
1603 Calhoun, Houston.
Curb, Dolph L., 6448 Fannin, Houston.
Curbo, James R., 3820 Fannin, Houston.
Curd, George W., Jr.,
2002 Holcombe Blvd., Houston.
Curtis, Dean D., 4205 Leeland Ave., Houston.
Daeschner, Chas. Wm., Jr.,
Baylor Med. School, Houston.
Dailey, James E.,
4109 Montrose Blvd., Houston.
Daily, Harold I.,
10521 S. Post Oak Rd., Houston.
Daily, Herschel, 4520 Ardennes St., Houston.
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6800 Long Point Rd., Houston.
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203 S. Munger Blvd., Pasadena.
Dargan, Joseph L.,
1737 W. Alabama, Houston.
Daughery, Jewel D., 5203 Valerie, Bellaire.
David, Solomon D.,
911 Med. Arts Bldg., Houston.
Davidson, Thomas H., 1820 W. 43rd, Houston.
Davis, Avolin Ross,
Hermann Prof. Bldg., Houston.
Davis, Charles Q., 2407 N. Main St., Houston.
Davis, Earl D.,
2255 W. Holcombe Blvd., Houston.
Davis, George W., Jr.,
1414 Hermann Prof. Bldg., Houston.
Davis, Glenn E., 201 N. 6th St., Bellaire.
Davis, Hamlet I., 9 North Ashbel, Baytown.
Davis, Phil,
1200 M. D. Anderson Blvd., Houston.
Davis, Robert M., Mem. Prof. Bldg., Houston.
Davis, Rufus C., Jr.,
906 E. Southmore, Pasadena.
Davis-Clark, Bertha M.,
1909 Sharp Pl., Houston.
Dawson, Donald C.,
906 E. Southmore, Pasadena.
Day, George P., (Inac.),
701 Med. Arts Bldg., Houston.
DeBakey, Michael E.,
1200 M. D. Anderson Blvd., Houston.
del Castillo, Hector,
4400 San Jacinto, Houston.
Denman, Frank R.,
309 Hermann Prof. Bldg., Houston.
Dennis, Edward W.,
1200 M. D. Anderson Blvd., Houston.
Derrick, William S.,
6723 Bertner Blvd., Houston.
Desmond, Murdina M.,
2210 Belfontaine, Houston.
DeTar, Webb T., Jr., (Int.),
3316 Norfolk, Apt. A, Houston.
DeVore, Marion S., 8777 Katy, Houston.
DeVore, Neal M.,
923 Med. Arts. Bldg., Houston.
Dew, Edward H., 5102 N. Main St., Houston.
DeYoung, Richard,
833 Hermann Prof. Bldg., Houston.
Diamond, Max M., 919 Maple, Pasadena.
Dickson, J. Charles,
Hermann Prof. Bldg., Houston.
Dinges, Malcolm D., Jr.,
6243 Old Richmond Rd., Houston.
Dinkler, Fred, 4310 Dunlavy St., Houston.
Dippel, A. Louis,
Hermann Prof. Bldg., Houston.
Dittman, Hyman, 1702 Heights Blvd., Houston.
Dittman, Sol L., 104 E. 20th St., Houston.
Divita, Eugene C.,
9935 Bessemer Dr., Houston.
Doak, Edmond K., Jr.,
Hermann Prof. Bldg., Houston.
Dobbs, Charles E.,
1616 Mem. Prof. Bldg., Houston.
Dobson, Harold L.,
Baylor U. College Med., Houston.
Dodd, George E., Jr., 5012 Travis, Houston.
Dodd, Gerald D., 6723 Bertner Dr., Houston.
Dodge, Warren F., (Int.),
5327 Dora, Apt. 1, Houston.
Dolph, Chancey H., Box 4281, Baytown.
Donald, Robert L., 411 W. 20th St., Houston.

Donnelly, Verner J.,
1100 Holcombe Blvd., Houston.
Donohue, William M., 6448 Fannin, Houston.
Donovan, Michael M.,
Hermann Prof. Bldg., Houston.
Dornak, Franklin K.,
4705 Montrose Blvd., Houston.
Dorsey, Frederick G.,
Hermann Prof. Bldg., Houston.
Doubleday, Leonard C.,
P. O. Box 7497, Houston.
Dougall, J. M.,
5000 Montrose Blvd., Houston.
Dougherty, Edward F.,
2708 Weslayan, Houston.
Drager, Glenn A.,
1200 M. D. Anderson Blvd., Houston.
Draper, Wm. Leonard,
2131 Brentwood, Houston.
Dross, Raymond L.,
1100 E. Holcombe Blvd., Houston.
DuCroz, James L., 617 Tatar, Pasadena.
Duff, Kenneth R., 2220 W. Alabama, Houston.
Duke, Herbert H., Box 359, Baytown.
Dunn, Ralph O., Jr.,
714 Med. Towers, Houston.
Durham, Charles A.,
411 W. 20th St., Houston.
Durham, Mylie E., 411 W. 20th St., Houston.
Durham, Mylie E., Jr.,
411 W. 20th St., Houston.
Duson, Craddock K., 4115 Fannin St., Houston.
Dustin, Herman E., 4400 San Jacinto, Houston.
Dutton, Robert V.,
7026 Stella Link Rd., Houston.
Duty, Rebecca T., 1811 Coral St., Houston.
Duty, Winthrop S., 1811 Coral St., Houston.
Dwyer, Charles A., Shamrock Hotel, Houston.
Earl, David M., Hermann Prof. Bldg., Houston.
Earlywine, Charles H., 8777 Katy Rd., Houston.
Eckles, Nylene E.,
M. D. Anderson Hosp., Houston.
Eckley, Robert,
Scott & White Clin., Temple.
Eden, George F., 1516 Pierce Ave., Houston.
Edwards, Robert A.,
307 Hermann Prof. Bldg., Houston.
Egan, Robert L.,
6723 Bertner Blvd., Houston.
Ehlers, H. Jack,
Hermann Prof. Bldg., Houston.
Ehni, George J., Hermann Prof. Bldg., Houston.
Eichelberger, Philip T., (Mil.),
3939 Southport St., San Antonio.
Eichhorn, Ralph D.,
1717 Med. Towers, Houston.
Eidman, Frederich G., 5644 Lawndale, Houston.
Ekman, C. J. Ivan, Med. Arts Bldg., Houston.
Elizondo, Cesar M.,
7428 Park Pl. Blvd., Houston.
Ellingson, Eugene A.,
1619 Med. Towers, Houston.
Elliott, Earlie E., 1401 Eagle, Houston.
Elliott, John J., 4400 San Jacinto, Houston.
Elliott, Monroe L., 4212 Caroline, Houston.
Elliott, Robert B.,
217 Med. Arts Bldg., Houston.
Embree, Elisha D., 2207 Stony Brook, Houston.
Emmert, Max W.,
345 Hermann Prof. Bldg., Houston.
Emmett, William B., 6003 Lyndhurst, Houston.
Engelhardt, Hugo T., Box 2180, Houston.
Engler, Joseph I., 104 Med. Towers, Houston.
Entzminger, Lindell B.,
2404 Rice Blvd., Houston.
Eppright, Richard H., 1803 Calumet, Houston.
Epstein, Isadore V., Box 429, Pasadena.
Erickson, Ethel E., V. A. Hospital, Houston.
Ernst, Frank J.,
Hermann Prof. Bldg., Houston.
Estess, Berthold H., 6215 Edloe, Houston.
Estrada, William J., 333 W. 20th St., Houston.
Etter, Richard L.,
156 Hermann Prof. Bldg., Houston.
Evans, Howard L.,
Hermann Prof. Bldg., Houston.
Everton, Marta Ve,
Hermann Prof. Bldg., Houston.
Fain, Robert H., 1803 Calumet St., Houston.
Fairchild, Robert J., 624 Tatar, Pasadena.
Farfel, Bernard, 1215 Med. Towers, Houston.
Faris, Arthur M., 2625 San Jacinto, Houston.
Farragut, Loyall D., Box 4116, Houston.
Fatherree, Thomas J., 6448 Fannin, Houston.
Faulkner, Hershell M.,
8917 Long Point Rd., Houston.
Fayle, Percy R., 2800 Garth Rd., Baytown.
Feamster, Robert C., 5202 Spruce, Bellaire.
Feinstein, Daniel J.,
1104 Med. Towers, Houston.
Felknor, George E., Jr.,
1004 W. Sterling, Baytown.
Ferguson, James P.,
11018 Atwell Dr., Houston.
Fernbach, Donald J., 6621 Fannin, Houston.
Ferwerda, Thomas,
703 Hermann Prof. Bldg., Houston.
Fields, William S.,
1200 M. D. Anderson Blvd., Houston.
Filippone, John M.,
801 Med. Arts Bldg., Houston.
Finch, William O., Jr.,
721 E. Texas Ave., Baytown.
Finney, R. Milton,
3701 Montrose Blvd., Houston.
Fiore, Charles N., 6725 Weslayan, Houston.
Fish, James R., 1204 Med. Towers, Houston.
Fishbein, Harry, 609 Med. Towers, Houston.
Fisher, William C., III,
Hermann Prof. Bldg., Houston.
Fitch, Edward A.,
309 Hermann Prof. Bldg., Houston.
Fitch, Edward O., 1729 Sunset Blvd., Houston.
Flanary, Lemuel M., Jr.,
530 W. 20th St., Houston.
Flanz, Bernard,
6303 South Park Blvd., Houston.
Fleet, Carl W., (dead), Houston.
Fletcher, Gilbert H.,
M. D. Anderson Hosp., Houston.
Fletcher, Mary W. C.,
906 Med. Towers, Houston.
Fletcher, Thomas A.,
2320½ Dowling, Houston.
Florence, Lewis, 7444 Harrisburg St., Houston.
Floyd, Osborne E.,
2100 Dowling at Gray, Houston.
Fly, Orceneth A., Jr.,
1212 Med. Towers, Houston.
Flynt, Otis P.,
1213 Med. Arts Bldg., Houston.
Foote, Stephen A., Jr.,
No. 2 Chelsea Pl., Houston.
Ford, Donald P.,
5000 Montrose Blvd., Houston.
Ford, George W., (Mil.),
322 Artemis Ave., San Antonio.
Ford, Ralph V., 5206 Richmond Ave., Houston.
Forde, George P. A., 618 Prairie, Houston.
Forister, Bryan W., Jr., (Int.),
3018 Lake, Apt. 8, Houston.
Forner, Vincent T., 809 Med. Towers, Houston.
Foster, John H., (Hon.),
700 S. McCarty Ave., Eagle Lake.
Foster, Juanita E., 219 W. Alabama, Houston.
Foster, Riley P.,
1512 Hermann Prof. Bldg., Houston.
Fountain, Edmund M.,
Hermann Prof. Bldg., Houston.
Frachtman, H. Julian,
2547 N. MacGregor Way, Houston.
Frankel, Richard A.,
5404 Palm Center, Houston.
Franklin, Robert R.,
1200 M. D. Anderson Blvd., Houston.
Fraser, Wilson M., (Int.),
5106 Grape Rd., Houston.
Frawley, John T., 1004 Seymour St., Pasadena.
Fredricks, Simon, 1103 Med. Towers, Houston.
Freeman, Bromley S.,
1810 Med. Towers, Houston.
Freeman, Robert G.,
1200 M. D. Anderson Blvd., Houston.
Freundlich, Charles G.,
2259 W. Holcombe, Houston.
Freundlich, Thomas W.,
3817 Fannin St., Houston.
Frey, C. Elmer, 2204 Baldwin St., Houston.
Friedman, David K.,
6303 S. Park Blvd., Houston.
Friedman, James A., (Int.),
1922 Wyndale, Apt. 2, Houston.
Friend, Victor V.,
1022 Med. Arts Bldg., Houston.
Frierson, James N., 1818 Caroline, Houston.
Fries, John G., (Int.),
Hermann Hospital, Houston.
Fromberg, Henry F., 6516 Bertner, Houston.
Fuller, Lillian M.,
6723 Bertner Blvd., Houston.
Gaitz, Charles M., 918 Med. Towers, Houston.
Gallager, H. Stephen,
M. D. Anderson Hosp., Houston.
Gamble, Jess F.,
814 Hermann Prof. Bldg., Houston.
Gamble, Jesse F., (Hon.),
401 Dennis St., Houston.
Gandy, D. Truett,
901 Med. Arts Bldg., Houston.
Gandy, Joe R., Hermann Prof. Bldg., Houston.
Gannon, John P.,
Hermann Prof. Bldg., Houston.
Gardner, Herman L., 6436 Fannin, Houston.
Gardner, Robert A.,
4006 Montrose Blvd., Houston.
Garrett, Edwin E.,
1616 Mem. Prof. Bldg., Houston.
Gaston, John Zell, (Hon.),
P. O. Box 147, Webster.
Gates, Charles S., Jr.,
So. Coast Life Bldg., Houston.
Gatoura, George J., 2404 Rice Blvd., Houston.
Gaulke, David W., 414 W. 20th, Houston.
Geibel, Roger S., 618 Tatar St., Pasadena.
Gemoets, Henry N.,
1506 Mem. Prof. Bldg., Houston.
Gemoets, Thomas H., (Int.),
1506 Mem. Prof. Bldg., Houston.
George, Gerald Y., 5214 Spruce, Bellaire.
Gerdes, Jack D., 7338 McHenry, Houston.
Gerdes, John R., Jr., (Int.),
5330 Windswept Lane, Houston.
Geringer, Benjamin J., 4901 Caroline, Houston.
Gibson, Fredrick J.,
222 Med. Arts Bldg., Houston.
Giessel, Julius W., 215 Milby, Houston.
Giessel, Lotta, 215 Milby, Houston.
Giessel, William U., 215 Milby, Houston.
Gillespie, Charles W.,
6800 Long Point Rd., Houston.
Girard, Louis J.,
Hermann Prof. Bldg., Houston.
Glantzberg, Herman,
Hermann Prof. Bldg., Houston.
Glass, Henry G.,
Hermann Prof. Bldg., Houston.
Glassman, Arthur L.,
Hermann Prof. Bldg., Houston.
Glen, John K., 4412 Montrose Blvd., Houston.
Glover, F. Scott, 4506 Caroline, Houston.
Goar, Everett L.,
1616 Mem. Prof. Bldg., Houston.
Godfrey, Edwin W., 602 Lamar, Houston.
Goens, Ray W., 6615 Travis, Houston.
Gol, Alexander,
1200 M. D. Anderson Blvd., Houston.
Gold, Jacob C.,
10229 Bauman Road, Houston.
Goldberg, Michael N.,
104 E. 20th St., Houston.
Gonzalez, Angel G.,
604 Scanlan Bldg., Houston.
Goodall, Robert J.,
1005 Med. Towers, Houston.
Goodloe, N. Maxwell,
1104 Hermann Prof. Bldg., Houston.
Goodrich, William A., Jr.,
P. O. Box 26, Baytown.
Goodwin, Roy T.,
4705 Montrose Blvd., Houston.
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Gordon, Wm. B., Jr.,
Hermann Prof. Bldg., Houston.
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6436 Fannin St., Houston.
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Hermann Prof. Bldg., Houston.
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Greene, James A.,
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Hermann Prof. Bldg., Houston.
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Greer, Cecil, 7438 Harrisburg Blvd., Houston.
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No. 2 Chelsea Pl., Houston.
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1200 M. D. Anderson Blvd., Houston.
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1805 Med. Towers, Houston.
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Grimm, John H., (Int.),
8715 Timberside, Houston.
Grininger, Donn Robert,
8003 Chocolate Bayou, Houston.
Grininger, George E., 1603 Lorraine, Houston.
Groff, A. Edward,
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Grunbaum, Franz V.,
1219 Med. Arts Bldg., Houston.
Guilford, Frederick R.,
Hermann Prof. Bldg., Houston.

Guin, Jere D., (Mil.),
3750th USAF Hosp., Sheppard AFB.
Guinn, Gene A.,
Hermann Prof. Bldg., Houston.
Gustafson, Robert E.,
4006 Montrose Blvd., Houston.
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Guy, E. Lloyd, Box 9786, Houston.
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M. D. Anderson Blvd., Houston.
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Haley, S. Willard,
511 Chronicle Bldg., Houston.
Hallson, D. C. McK.,
5506 Russett Dr., Houston.
Halpert, Bela,
2002 Holcombe Blvd., Houston.
Ham, Goldie S., 2708 Weslayan, Houston.
Hamilton, Carlos R., 5212 Caroline, Houston.
Hamrick, Wendell H., 218 Main St., Houston.
Haney, Fred T., 3724 Broadway, Houston.
Hankins, Lawson A., 119 W. Defee, Baytown.
Hannon, Theo R., 2625 San Jacinto, Houston.
Hansen, William M.,
8003 Chocolate Bayou, Houston.
Hanson, Hugh H., 6448 Fannin, Houston.
Hanson, Leroy W., 10 Chelsea Pl., Houston.
Harberg, Franklin J.,
1702 Med. Towers, Houston.
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Hermann Prof. Bldg., Houston.
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Harris, T. Fred, Med. Arts Bldg., Houston.
Harrison, Gunyon M.,
1333 Moursund Ave., Houston.
Harrison, Malcolm W.,
1507 Med. Arts Bldg., Houston.
Harrison, William T., 5400 LaBranch, Houston.
Hart, George H., 5608 Yale St., Houston.
Hart, Juanita T., 5620 Flack Dr., Houston.
Hartgraves, Ruth, 1208 Med. Towers, Houston.
Harwell, William S., 602 Lamar, Houston.
Harwood, Nathan, 714 Med. Towers, Houston.
Haufrect, Fred, 806 Med. Towers, Houston.
Haus, Loren W., 4832 Caroline Ave., Houston.
Hauser, Abe, 1119 Lovett Blvd., Houston.
Hauser, Harris M., (Int.),
1515 Graham Ct., Rochester, Minn.
Hawkins, Theron C., (Int.),
2807 Tangley Rd., Houston.
Hay, Elliott B., P. O. Box 9836, Houston.
Hay, Richard C.,
6723 Bertner Blvd., Houston.
Hayes, Herbert T.,
636 Hermann Prof. Bldg., Houston.
Haywood, Theodore J.,
3509 Montrose Blvd., Houston.
Headrick, Charles B., 2136 Yale St., Houston.
Healey, John E., Jr.,
6723 Bertner Dr., Houston.
Heard, J. Griffin,
Hermann Prof. Bldg., Houston.
Heisler, John J., 6417 Richmond Rd., Houston.
Helman, Rowland J., 1010 Banks St., Houston.
Hendry, Cullen H., (Inac.),
2332 Wordsworth, Houston.
Henly, Walter S.,
4330 Kingfisher St., Houston.
Henry, Marshall G.,
Hermann Prof. Bldg., Houston.
Hensley, Barnes C., 1111 Evergreen, Houston.
Herman, Julius W.,
721 E. Texas Ave., Baytown.
Hershberger, Robt. L.,
5410 Palms Center, Houston.
Hesse, Clyde E., 9935 Bessemer St., Houston.
Hethcock, Lewis D., (Mil.), Rt. 1, LaFeria.
Hettig, Robert A.,
1200 M. D. Anderson Blvd., Houston.
Higginbotham, Warren H.,
264 Hermann Prof. Bldg., Houston.
Higgins, Clarence R., Jr.,
5101 Lockwood Dr., Houston.
Hild, Jack R., 4414 Montrose Blvd., Houston.
Hill, Austin E., 1300 Capitol Ave., Houston.
Hill, Beth M., 10401 Palestine, Houston.
Hill, Charles A., 810 Caroline St., Houston.
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1729 Sunset Blvd., Houston.
Hill, Jasper H., (Hon.), Bandera.
Hill, John R., (Int.),
1029 Kirby Dr., Houston.
Hill, L. Leighton,
1200 M. D. Anderson Blvd., Houston.
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Hill, Robert M.,
Hermann Prof. Bldg., Houston.
Hill, William T., 6621 Fannin St., Houston.
Hinds, Edward C., 1508 Med. Towers, Houston.
Hinds, Gordon F., 2003 San Jacinto, Houston.
Hines, Norman D., 2317 Fannin, Houston.
Hinterlang, Victor A.,
1024 Walling St., Houston.
Hinton, Leslie, Box 311, Crosby.
Hoaglin, Lester L., Jr.,
3701 Montrose Blvd., Houston.
Hodell, George R., 2418 Travis, Houston.
Hodges, Lynn P., Box 3086, Baytown.
Hoeflich, Emilia F. A.,
1516 Pierce Ave., Houston.
Hofer, Jesse W.,
107 Hermann Prof. Bldg., Houston.
Hoffmann, Clifford J., (Int.),
11617 Blalock Forest, Houston.
Holland, Theo L., 1018 Richmond, Houston.
Holle, Henry B., (Int.),
3210 Maroneal, Houston.
Hollimon, James H.,
P. O. Box 18246, Houston.
Hollins, Blanchard T.,
5101 Lockwood, Houston.
Holloman, Walter G.,
Hermann Prof. Bldg., Houston.
Holloran, Richard J.,
916 Bank of Southwest, Houston.
Holloway, Dan W., 4214 Caroline, Houston.
Hollub, Charles J.,
711 Med. Arts Bldg., Houston.
Holsomback, J. C., 119 W. Defee, Baytown.
Hooker, Lyle, Mem. Prof. Bldg., Houston.
Hooper, Christopher C.,
1029 E. Thomas St., Pasadena.
Hoovestol, Paeder J., 6436 Fannin St., Houston.
Hotchkiss, DeWitt H., Jr.,
Hermann Prof. Bldg., Houston.
Howard, A. Philo, (Hon.),
4119 Montrose Blvd., Houston.
Howe, Clifton D., 6723 Bertner, Houston.
Howell, Theodore S.,
1601 California, Baytown.
Hucherson, Denman C.,
Hermann Prof. Bldg., Houston.
Hudgins, Thomas F., Jr.,
1204 Med. Towers, Houston.
Hughes, Louis B.,
119 W. Defee Ave., Baytown.
Hughes, William E.,
906 E. Southmore, Pasadena.
Hull, John G., 3701 Montrose Blvd., Houston.
Humphrey, Stanley G.,
1921 W. Main St., Baytown.
Hundley, Richard Z., 1010 Banks St., Houston.
Hunter, Malcolm E., Jr.,
1101 W. Lobit St., Baytown.
Hutcheson, Allen C., (Hon.),
1720 Milford, Houston.
Hutchins, Selwyn P. R.,
1232 Hermann Prof. Bldg., Houston.
Iannucci, Chriss A.,
2259 W. Holcombe, Houston.
Ibanez, Michael L.,
6723 Bertner Ave., Houston.
Iiams, Frank J., (Hon.), 2204 Troon, Houston.
Izen, Joe A., 704 Tatar St., Pasadena.
Jachimczyk, Joseph A.,
209 Harris Co. Courthouse, Houston.
Jackson, Daniel, 810 Med. Towers, Houston.
Jackson, R. Andrew, (Int.),
4325 Cynthia, Bellaire.
Jackson, Richard H.,
156 Hermann Prof. Bldg., Houston.
Jackson, Robert H.,
1516 Pierce St., Houston.
Jackson, Robert R., Jr.,
1801 Buffalo Dr., Houston.
Jackson, Thomas F.,
1101 Med. Arts Bldg., Houston.
Jacobs, Warren M.,
1009 Med. Towers, Houston.
Jagoda, Samuel, Jr., (Int.),
1501 Summit, Fort Worth.
Jansa, Arthur M., 2038 MacArthur, Houston.
Jansa, Frances H., 503 Med. Towers, Houston.
Janse, H. M., 2010 Crawford St., Houston.
Jaubert, Francis L., Jr.,
1004 W. Sterling, Baytown.
Jenkins, Daniel E.,
1200 M. D. Anderson Blvd., Houston.
Jenkins, Edward E., 4822 Linden, Bellaire.
Jensen, Francine, 2218 W. Main St., Houston.
Jerabeck, John D., 2006 W. Alabama, Houston.
Jesse, Richard H., 6723 Bertner Ave., Houston.
Jester, Albert W., Rt. 1, Box 306, Crosby.
Joachim, Harold S.,
1820 W. 43rd St., Houston.
Johnson, Craig B.,
5000 Montrose Blvd., Houston.
Johnson, Gordon C.,
6723 Bertner Blvd., Houston.
Johnson, Joseph K., II,
7338 McHenry, Houston.
Johnson, Lawrence W.,
6704 Capitol Ave., Houston.
Johnson, Marilyn, 4510 Caroline St., Houston.
Johnson, R. Marion,
Hermann Prof. Bldg., Houston.
Johnson, Seale I.,
2521 Robinhood St., Houston.
Johnston, Robert A., 4115 Fannin, Houston.
Jones, Bobby, 218 Main St., Houston.
Jones, Corinne C. M.,
3207 Montrose Blvd., Houston.
Jones, Franklyn,
8121 Chocolate Bayou Rd., Houston.
Jones, J. Randolph,
1708 Med. Towers, Houston.
Jones, James C.,
Hermann Prof. Bldg., Houston.
Jones, J. Sid,
1101 Hermann Prof. Bldg., Houston.
Jones, Malcolm A., 119 W. Defee, Baytown.
Jones, Peron O., 1701 S. Shepherd, Houston.
Jones, Roy V.,
3207 Montrose Blvd., Houston.
Jones, S. Ross, (Hon.), Box 773, Portland.
Jones, Tom R.,
1200 S. Shepherd, Houston.
Jones, William P., Jr.,
906 E. Southmore, Pasadena.
Jones, Willis T., Jr.,
2800 Garth Rd., Baytown.
Jordan, George L., Jr.,
2002 Holcombe Blvd., Houston.
Jorns, C. Forrest, 5644 Lawndale, Houston.
Joseph, Sheldon W.,
6303 S. Park Blvd., Houston.
Kahle, Warren F.,
4705 Montrose Blvd., Houston.
Kaiser, Clarence H.,
6800 Long Point Rd., Houston.
Kalb, Theodore W.,
418 Med. Arts Bldg., Houston.
Kaminsky, Dave, 7117 Lyons, Houston.
Kantis, John G., 3803 Harrisburg, Houston.
Kaplan, Harry L., 6448 Fannin, Houston.
Karbach, Nelson W., Jr.,
410 W. 20th St., Houston.
Karnaky, Karl J., 2614 Crawford, Houston.
Karotkin, Lester,
Hermann Prof. Bldg., Houston.
Kastleman, Jerry Lee,
10521 S. Post Oak Rd., Houston.
Katribe, Paul, 7639 Long Point Rd., Houston.
Kaufman, Raymond H.,
201 N. 6th St., Bellaire.
Kay, Jacob L.,
1200 M. D. Anderson Blvd., Houston.
Kearns, Edward E., 7122 Hartland, Houston.
Keats, Arthur S., 2401 Dryden Rd., Houston.
Keegan, James M.,
616 Med. Arts Bldg., Houston.
Kelsey, John R., Jr., 6655 Travis, Houston.
Kelsey, Mavis P., 6655 Travis, Houston.
Kemper, James W., 6655 Travis St., Houston.
Kendall, Dean H.,
226 Med. Arts Bldg., Houston.
Kennedy, John C.,
Hermann Prof. Bldg., Houston.
Kennerly, Thomas P.,
8849 Long Point Rd., Houston.
Kent, Glen R., P. O. Box 726, Channelview.
Kerr, C. Denton,
Hermann Prof. Bldg., Houston.
Kerr, Wm. Rupert, 6436 Fannin, Houston.
Kilgore, F. Hartman,
1118 Eagle Ave., Houston.
Kilgore, Morris W.,
1718 Med. Towers, Houston.
Kilgore, Newton A.,
4119 Montrose Blvd., Houston.
Kilpatrick, Hamilton W.,
1701 Oak Shadows, Baytown.
Kincaid, Harvey L.,
5400 La Branch St., Houston.
King, Joe W., 1803 Calumet St., Houston.
Kinross-Wright, V. J.,
1200 M. D. Anderson Blvd., Houston.
Kirkham, Sam H., 2807 Little York, Houston.
Kirkland, Arthur A., 2701 Berry Rd., Houston.
Kirkpatrick, Robert H.,
5150 Jackwood, Houston.
Kirkpatrick, Wm. E., 2811 Plumb St., Houston.

Kirschner, Rudolf, (Mil.),
101 E. Western Ave., Avondale, Ariz.
Kitchen, Benjamin F., Jr.,
10 Chelsea Place, Houston.
Klanke, Charles W.,
4040 Broadway Blvd., Houston.
Klein, Perry B., 709 Med. Towers, Houston.
Kline, Allen H., (Int.),
517 Flintdale, Houston.
Kneip, A. T., (Hon.),
940 Teeshorn, Houston.
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3407 Montrose Blvd., Houston.
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Knox, John M.,
1200 M. D. Anderson Blvd., Houston.
Koch, Herbert E., 607 Witcher Lane, Houston.
Koenig, Frank J.,
4807 Montrose Blvd., Houston.
Kolaja, John W., Box 875, Almeda.
Kolodny, George R.,
1217 Med. Towers, Houston.
Kolter, Wm. Henry, Jr., (Int.),
Hermann Hospital, Houston.
Korndorffer, Wm. E., Jr., (Mil.),
5505 Dennis St., Fort Worth.
Kraft, Irvin A., 2310 Baldwin, Houston.
Kubala, Mark J., (Int.),
Hermann Hosp., Houston.
Kuebler, Luke W.,
1403 Med. Arts Bldg., Houston.
LaGrone, Columbus L.,
8706 Pritchett St., Houston.
Lambert, Y. Alan, Jr.,
1614 Holland St., Houston.
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1729 Sunset Blvd., Houston.
Langford, C. H., (Hon.), Bandera.
Lapat, William, (Hon.),
2715 Arbuckle, Houston.
Latimer, Mark H.,
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519 Medical Towers, Houston.
Lattimore, Joseph S.,
1004 Hermann Prof. Bldg., Houston.
Lauden, Alfred E.,
1405 Hermann Prof. Bldg., Houston.
Laurentz, Fred K., Houston City Hall, Houston.
Laurie, Ben Erle, 12748 Bertha Lane, Houston.
Law, Samuel W.,
2002 Holcombe Blvd., Houston.
Lawrence, Buell A.,
4205 Leeland Ave., Houston.
Lawrence, Van L.,
907 Hermann Prof. Bldg., Houston.
Lawrence, Wilbert, 2947 Dragonwick, Houston.
Leach, Charles L.,
358 M & M Bldg., Houston.
Leachman, Robert D.,
519 Med. Towers, Houston.
Leader, Abel J.,
1813 Med. Towers, Houston.
Leary, William V., 6655 Travis, Houston.
Leaton, Robert E.,
4414 Montrose Blvd., Houston.
Leavens, Milam E.,
1006 Hermann Prof. Bldg., Houston.
Ledbetter, Abbe A.,
1603 Med. Arts Bldg., Houston.
Ledbetter, Paul V.,
1603 Med. Arts Bldg., Houston.
Lee, William L., Jr., (Mil.),
123 Montpeliar, San Antonio.
LeGall, Fitzherbert H.,
222 West Dallas, Houston.
Leggett, Milbourne K.,
5000 Montrose Blvd., Houston.
Leidler, Franz,
1201 Mem. Prof. Bldg., Houston.
Leifeste, Homer F.,
Hermann Prof. Bldg., Houston.
Leigh, Richard E., Jr.,
1613 Med. Towers, Houston.
Leiser, Alfred E., 6655 Travis St., Houston.
Lemak, Leslie L., 1004 Med. Towers, Houston.
Lensky, Paul, 105 Med. Towers, Houston.
Leong, Richard W., 1817 Dryden Rd., Houston.
LePere, Donald M.,
5504 Irvington Blvd., Houston.
Lerner, Ben L., 3817 Fannin St., Houston.
Levin, Louis, 7521 Humble Rd., Houston.
Levine, Isaac, (Int.),
6516 Bertner Dr., Houston.
Levy, George, 8561 Long Point Rd., Houston.
Levy, Jaime, 6804 Ave. N, Houston.
Levy, Moise D., 3618 Yoakum Blvd., Houston.
Levy, Moise D., Jr.,
3618 Yoakum Blvd., Houston.
Levy, Samuel A., 605 Med. Towers, Houston.
Lewis, Arthur N.,
Hermann Prof. Bldg., Houston.
Lewis, Everett B.,
Hermann Prof. Bldg., Houston.
Lewis, L. Roy, 410 W. 20th St., Houston.
Lewis, Noel R., (Int.),
Hermann Hospital, Houston.
Leyva, Angel, 1510 Quitman St., Houston.
Lhamon, William T.,
1200 M. D. Anderson Blvd., Houston.
Lieppman, Jack E.,
414 Med. Arts Bldg., Houston.
Liggett, Charles Lee, 1015 Polly St., Baytown.
Ligon, Joseph G., (Hon.),
2214 Dorrington Ave., Houston.
Liles, Ralph, 1818 Caroline, Houston.
Lind, Carl J., Jr., 6720 Bertner Dr., Houston.
Lindsey, Bert G., Jr., 5200 Spruce St., Bellaire.
Lipscomb, Harry S.,
1200 M. D. Anderson Blvd., Houston.
Littell, Milton, 6521 Harrisburg, Houston.
Little, Harry M., 304 McIlhenny St., Houston.
Livesay, William R.,
4109 Montrose Blvd., Houston.
Logue, Lyle J.,
1600 Mem. Prof. Bldg., Houston.
Lomas, Robert D., 1731 Sunset Blvd., Houston.
Lorenzen, Evelyn J.,
3911 Montrose Blvd., Houston.
Lowe, Percy E., Mem. Prof. Bldg., Houston.
Lowe, Thomas E.,
P. O. Box 18246, Houston.
Lucas, J. Beeman, 410 W. 20th St., Houston.
Lucci, Joseph A., Jr.,
703 Med. Towers, Houston.
Lundgren, Lawrence E.,
8363 Winkler Dr., Houston.
Lunin, Arthur B., 4003 Bellaire Blvd., Houston.
Lyons, Robert D., 4205 Leeland Ave., Houston.
MacComb, William S.,
6723 Bertner Blvd., Houston.
MacIntyre, Robert S.,
Hermann Prof. Bldg., Houston.
Madison, John C., 2801 Blodgett, Houston.
Madsen, Alva C.,
7444 Harrisburg Blvd., Houston.
Magee, Mary Lucille,
1811 Crawford St., Houston.
Malewitz, Edward C.,
3618 Yoakum Blvd., Houston.
Mallerich, Melvin J., 1516 Pierce St., Houston.
Maness, Gerald W., 414 W. 20th St., Houston.
Mannheimer, Ilse H.,
6723 Bertner Dr., Houston.
Marcuse, Peter M., St. Josephs Hosp., Houston.
Maresh, Rudolph E.,
4520 Rossmoyne St., Houston.
Margraves, Ross D.,
Hermann Prof. Bldg., Houston.
Markewich, Jake, 1305 Blodgett, Houston.
Marshall, Reagan M., 3207 Fannin, Houston.
Marshall, William E., Box 3150, Baytown.
Martin, Cayo A., 906 Wayside, Houston.
Martin, Clarence R.,
1426 Med. Arts Bldg., Houston.
Martin, John E.,
616 Med. Arts Bldg., Houston.
Martin, Richard G.,
M. D. Anderson Hosp., Houston.
Mathews, William A., 624 S. Tatar, Pasadena.
Mathis, Kenneth G.,
906 E. Southmore, Pasadena.
Mathis, Robert L.,
3403 Montrose Blvd., Houston.
Maxwell, Jay C.,
104 Medical Towers, Houston.
Mayes, Lee P., 618 Tatar St., Pasadena.
Mayfield, Jack H.,
Hermann Prof. Bldg., Houston.
Mazow, Jack B., 605 Med. Towers, Houston.
McAmis, Robert J., 1210 Uvalde St., Houston.
McCallum, Marion J.,
2701 Berry Rd., Houston.
McCartney, Gene W., 201 N. 6th St., Bellaire.
McCarver, Charles T., 2012 Crawford, Houston.
McCasland, Clifford, 125 S. Main, Highlands.
McClanahan, H. Lane,
2807 S. Braeswood, Houston.
McCleery, James M.,
1004 Seymour St., Pasadena.
McClure, Robert A.,
4426 Tonawanda St., Bellaire.
McConn, Robert G.,
504 Hermann Prof. Bldg., Houston.
McConnell, Jesse M.,
5504 Irvington Blvd., Houston.
McCulley, J. D., 2334 Tangley Rd., Houston.
McDonald, James F., Jr.,
354 Hermann Prof. Bldg., Houston.
McDonald, Newton F.,
915 Med. Arts Bldg., Houston.
McElveen, William C.,
8762 Long Point Rd., Houston.
McGehee, Frank O., 5220 Travis, Houston.
McGivney, Felix F., Jr.,
530 Hermann Prof. Bldg., Houston.
McGovern, John P.,
3509 Montrose Blvd., Houston.
McGrath, Warren J.,
10521 S. Post Oak Rd., Houston.
McGraw, John P.,
3503 Montrose Blvd., Houston.
McGuire, John O., 2701 Berry Rd., Houston.
McGuire, Thomas H.,
601 Med. Arts Bldg., Houston.
McGurl, Frank J.,
1100 Holcombe Blvd., Houston.
McHenry, R. Kingsley, Jr., (Int.),
3660 Chevy Chase, Houston.
McIndoe, Frank W.,
1509 Med. Arts Bldg., Houston.
McKay, Haden E., Jr., Box 625, Humble.
McKinley, Thomas R.,
1108 Houston Blvd., South Houston.
McKinney, Mary Ann,
5000 Montrose Blvd., Houston.
McMeans, Robert H.,
1201 Med Arts Bldg., Houston.
McMullen, Faber F., Jr., (Int.),
Hermann Hosp., Houston.
McMurrey, Allen L., 1204 Eagle St., Houston.
McMurrey, James D.,
1200 M. D. Anderson Blvd., Houston.
McMurrey, Samuel P.,
1000 East James St., Baytown.
McNamara, Dan G., 6621 Fannin St., Houston.
McNatt, James K., 2701 Berry Rd., Houston.
McNeely, John F., Box 872, Pasadena.
McNeill, A. S., 5301 Fannin St., Houston.
McPeak, Edgar M.,
4109 Montrose Blvd., Houston.
McPherson, Alice R.,
1131 Hermann Prof. Bldg., Houston.
McReynolds, Isaac S.,
815 Med. Arts Bldg., Houston.
McReynolds, John W., Jr.,
5608 Yale St., Houston.
McReynolds, Robert J.,
5822 Broadway, Houston.
McRoberts, William A.,
Hermann Prof. Bldg., Houston.
McSpadden, Floyd F.,
533 W. 20th St., Houston.
Meisler, Irwin M., 5217 Holmes Rd., Houston.
Melton, Walter T.,
636 Hermann Prof. Bldg., Houston.
Meltzer, Leonard, 1413 Med. Towers, Houston.
Mendell, David, 1706 Med. Towers, Houston.
Mendell, Henry E.,
4003 Bellaire Blvd., Houston.
Mendell, Jack L., 4003 Bellaire Blvd., Houston.
Merriman, George J., Jr.,
P. O. Box 14464, Houston.
Messer, Jesse N., Med. Arts Bldg., Houston.
Messerschmidt, H. C., Jr.,
1009 Polly St., Baytown.
Meyer, Henry S., 1203 Cleburne, Houston.
Meynier, Maurice J., Jr.,
1601 Calhoun, Houston.
Milani, Rodolfo E., Jeff Davis Hosp., Houston.
Miller, A. Pidd, 6135 Cavanaugh St., Houston.
Miller, Arthur L., (Inac.),
1317 Yale St., Houston.
Miller, G. Maxwell,
202 San Jacinto St., LaPorte.
Miller, George V.,
165 Hermann Prof. Bldg., Houston.
Miller, Gus, 104 E. 20th St., Houston.
Miller, Howard E.,
1704 Crawford St., Houston.
Miller, Lowell S.,
6723 Bertner Blvd., Houston.
Miller, William A.,
1010 N. Federal Rd., Houston.
Milligan, John J.,
4702 Yoakum Blvd., Houston.
Mills, Morriss, Jr.,
2224 Bellefontaine, Houston.
Minor, Walter J., 3024 Holman St., Houston.
Mintz, A. Aaron,
1318 Telephone Rd., Houston.
Mitchell, A. Lane, 3820 Fannin St., Houston.
Mitchell, Donald D., Box 3428, Bellaire.
Mitchner, James M.,
412 W. 32nd St., Houston.
Mock, Presley J., P. O. Box 638, LaPorte.
Modisette, Henry C.,
1324 Federal Rd., Houston.
Moers, Arthur E., 401 Melrose Bldg., Houston.
Moers, Robert O.,
1325 Med. Arts Bldg., Houston.
Mohle, Flavius D.,
3902 Montrose Blvd., Houston.
Moise, Leonard D.,
509 Indiana, South Houston.

Moldawer, Marc,
1200 M. D. Anderson Blvd., Houston.
Montague, Laura J., 6613A Travis, Houston.
Montague, Meredith, III,
4705 Montrose, Houston.
Montgomery, C. Hunter,
Hermann Prof. Bldg., Houston.
Montgomery, Charles F.,
301 Med. Arts Bldg., Houston.
Montgomery, Hal E.,
5124 Old Richmond Rd., Bellaire.
Montgomery, Johnny L., 211 S. Main, Pasadena.
Moody, Irving W., 2418 Travis St., Houston.
Moody, Joe Marshall, 602 Lamar St., Houston.
Moore, Conard D., (Int.),
5707 H.M.C. St., Apt. 7, Houston.
Moore, Duncan L.,
Hermann Prof. Bldg., Houston.
Moore, George L., 709 Med. Towers, Houston.
Moore, Jack, Hermann Prof. Bldg., Houston.
Moore, John M., Jr.,
7444 Harrisburg, Houston.
Moore, Robert A., 1106 Med. Towers, Houston.
Moore, Thomas O., Jr.,
Hermann Prof. Bldg., Houston.
Moorhead, William H.,
5000 Montrose Blvd., Houston.
Moorman, Jesse A., Jr.,
13131 Rockglen Dr., Houston.
Morgan, Ray M., Jr.,
Hermann Prof. Bldg., Houston.
Morris, Al Kelly, 1001 Polly, Baytown.
Morris, George C., Jr.,
1200 M. D. Anderson Blvd., Houston.
Morrison, J. Winston,
4528 Griggs Rd., Houston.
Morrow, Edwin J.,
1603 Med. Arts Bldg., Houston.
Moten, William M.,
300 Hermann Prof. Bldg., Houston.
Moursund, Myles P.,
1019 Hermann Prof. Bldg., Houston.
Mumford, David M.,
6154 Westheimer, Houston.
Myers, Claude D., (Inac.),
2104 Pelham Dr., Houston.
Nail, Ben M., Jr., 1002 Med. Towers, Houston.
Nance, John O., 325 W. 20th St., Houston.
Neidhardt, Heinrich W.,
M. D. Anderson Hosp., Houston.
Neil, Carleton M.,
5206 Richmond Ave., Houston.
Nelson, Norman C., (Int.),
Charity Hosp., New Orleans, La.
Nelson, Robert S.,
6723 Bertner Blvd., Houston.
Nesselrode, John E., 3130 Wisconsin, Baytown.
Nettles, Mark K., 4604 McCarty St., Houston.
Neumaier, Hilde, 1010 Federal Rd., Houston.
Newton, Berne L.,
Methodist Hospital, Houston.
Nichols, John Wm.,
208 North Main, Galena Park.
Nickey, Laurance N., (Mil.),
901 Cincinnati St., El Paso.
Nicosia, Ralph V., 805 Westheimer, Houston.
Norris, John E., 6448 Fannin, Houston.
Norris, Ronald F.,
1610 Mem. Prof. Bldg., Houston.
Norton, Joseph G., 4205 Leeland, Houston.
Nunnally, Richard M.,
6516 Bertner Blvd., Houston.
Oates, James R.,
615 Hermann Prof. Bldg., Houston.
O'Brien, Richard F., 1713 Huge Oaks, Houston.
O'Brien, Robert B., 1713 Huge Oaks, Houston.
O'Donnell, William W.,
3200 Caroline, Houston.
O'Heeron, Michael K.,
1804 Med. Towers, Houston.
Ohlhausen, S. G., 2827 Renshaw, Houston.
Oldham, Dudley Y., 5503 Crawford, Houston.
Olin, Walter G., Jr.,
165 Hermann Prof. Bldg., Houston.
Oliver, J. Stanley, 7444 Harrisburg, Houston.
Oliver, John T., 7444 Harrisburg, Houston.
Oliver, William D., (Inac.),
Box 61, Galena Park.
Olsen, Roy W., 6243 Richmond Rd., Houston.
Olson, Stanley W.,
1200 M. D. Anderson Blvd., Houston.
Orman, McDonald,
925 Med. Arts Bldg., Houston.
Orr, Guy H., 5043 Bellfort, Bellaire.
Ory, Edwin M., 810 Med. Towers, Houston.
Osborne, Clarence F., Box 769, Pasadena.
Overstreet, John W.,
Hermann Prof. Bldg., Houston.
Overton, Robert C., Jr.,
Hermann Prof. Bldg., Houston.
Owen, William R.,
Hermann Prof. Bldg., Houston.
Owens, John B., 613 Harold Ave., Houston.
Owers, Albert, 6655 Travis St., Houston.
Owsley, William C., Jr.,
214 Hermann Prof. Bldg., Houston.
Padilla, Guillermo, 2502 Canal St., Houston.
Page, J. Herbert, (Inac.),
5517 Crawford St., Houston.
Painter, Joseph T.,
1603 Med. Arts Bldg., Houston.
Palm, William M., 2014 Crawford, Houston.
Panzarella, Carlo J., P. O. Box 766, Humble.
Park, David R.,
4010 Montrose Blvd., Houston.
Parker, George E., 4115 Caroline, Houston.
Parr, Luther H., 3609 Elmridge, Houston.
Parrish, Frank F.,
Hermann Prof. Bldg., Houston.
Paton, Donald M., 6611 Travis, Houston.
Patrick, Ralph C.,
907 Med. Arts Bldg., Houston.
Patten, Thelma A., 222 W. Dallas St., Houston.
Patterson, Robert T., 105 West 9th St., Mission.
Pawelek, I. L., (Inac.),
2211 Augusta Dr., Houston.
Pawelek, Louis G.,
222 Med. Arts Bldg., Houston.
Peck, T. T., Jr., 721 E. Texas Ave., Baytown.
Peddie, George H.,
1205 Hermann Prof. Bldg., Houston.
Pemberton, Charles W.,
222 W. Dallas St., Houston.
Pentecost, Lewis V., 208 N. Main, Galena Park.
Perdue, George W., No. 2 Chelsea Pl., Houston.
Perry, John L., Jr., 4705 Montrose, Houston.
Peters, I. D., Hermann Prof. Bldg., Houston.
Petersen, Henry A.,
820 Med. Arts Bldg., Houston.
Peterson, Carl A., 4710 Lillian, Houston.
Petri, Karin A., 4119 Montrose Blvd., Houston.
Petway, Aileen, 3302 Cloverdale St., Houston.
Petway, Max E., 8802 Curry Rd., Houston.
Pfeffer, John F., 1516 Pierce, Houston.
Pfeiffer, Eva Marie,
5404 Palms Center, Houston.
Phillips, Charles, 3745 Arnold Ave, Houston.
Phillips, Elliott S., 1803 Crawford, Houston.
Phillips, John M., Jr., 6448 Fannin, Houston.
Phillips, John R., Med. Arts Bldg., Houston.
Phillips, Leon, 1902 Blodgett, Houston.
Phipps, John,
8262 Park Pl. Blvd., Houston.
Pickett, Lee Lloyd,
P. O. Drawer 2100, Houston.
Pipkin, Robert W., Box 3663, Baytown.
Pittman, James E.,
622 Hermann Prof. Bldg., Houston.
Pokorny, Alex D.,
2002 Holcombe Blvd., Houston.
Pope, James W.,
Hermann Prof. Bldg., Houston.
Potts, Charles R.,
1616 Mem. Prof. Bldg., Houston.
Powell, Elizabeth B.,
2129 Brentwood, Houston.
Powell, Norborne B.,
830 Hermann Prof. Bldg., Houston.
Poyner, Herbert F.,
1014 Med. Arts Bldg., Houston.
Pranke, Don W.,
411 Hermann Prof. Bldg., Houston.
Pratt, Helen L., 7012 S. 7th St., Houston.
Pratt, Willard M., 918 Lovett, Houston.
Price, William P., 1009 W. Main St., Houston.
Pritchett, I. E., (Hon.), Angleton.
Pronin, Arthur,
3004 Little York Rd., Houston.
Pruitt, Raymond D.,
1200 M. D. Anderson Blvd., Houston.
Pugsley, Cornelius, Jr.,
Hermann Prof. Bldg., Houston.
Purnell, Guy W., 411 W. 20th St., Houston.
Purnell, Howard G.,
8916 Spring Branch Dr., Houston.
Qualtrough, Walter F.,
4716 Montrose Blvd., Houston.
Radcliffe, Glen J., 10805 Willowisp, Houston.
Radding, Robert S., 6615 Travis, Houston.
Ramsay, William E.,
4705 Montrose Blvd., Houston.
Rape, Marvin G.,
915 Med. Arts Bldg., Houston.
Raymer, Warren J.,
156 Hermann Prof. Bldg., Houston.
Redmon, Agile H., Jr.,
Hermann Prof. Bldg., Houston.
Rednick, Meyer S., 104 E. 20th St., Houston.
Reece, Charles D., 6448 Fannin, Houston.
Reed, Joel E., 6448 Fannin St., Houston.
Reinke, R. Thomas, 2708 Weslayan, Houston.
Renfrow, W. Frank,
1407 Mem. Prof. Bldg., Houston.
Ribelles, Jacqueline L.,
985 Bunker Hill Rd., Houston.
Ribelles, Jose A.,
985 Bunker Hill Rd., Houston.
Richardson, Joe B.,
1217 Hermann Prof. Bldg., Houston.
Ried, Hubert L., 2229 Dorrington, Houston.
Riley, Gorman L., 4515 Mangum Rd., Houston.
Rivers, Franklin M.,
350 Hermann Prof. Bldg., Houston.
Robbins, E. Freeman,
2024 Richmond Ave., Houston.
Robbins, Horace T.,
2024 Richmond Ave., Houston.
Robbins, Leonard R.,
3618 Yoakum Blvd., Houston.
Roberson, Edward, Jr., 1210 Uvalde, Houston.
Roberts, Newell O.,
1701 S. Shepherd, Houston.
Robertson, Joseph W.,
Hermann Prof. Bldg., Houston.
Robertson, Robt. C. L.,
Hermann Prof. Bldg., Houston.
Robey, Grace Lucile,
802 Hermann Prof. Bldg., Houston.
Robey, Louis R., 4104 Lyons Ave., Houston.
Robinett, James B., Jr., 3434 Norris, Houston.
Robins, Bill, Continental Bank Bldg., Houston.
Robinson, Hampton C.,
Hermann Prof. Bldg., Houston.
Robinson, W. Tryon, 6410 Fannin, Houston.
Robison, J. M.,
1600 Mem. Prof. Bldg., Houston.
Robison, Jehu M., Jr., (Int.),
2303 Midlane, No. 286, Houston.
Rodgers, Lawrence R.,
5000 Montrose Blvd., Houston.
Roett, Catherine J. E.,
3262 Holman St., Houston.
Rogers, Stanley F., 1009 Med. Towers, Houston.
Rohrer, Geo. E.,
1110 Med. Arts Bldg., Houston.
Rose, Jack M., Hermann Prof. Bldg., Houston.
Rose, Raymond G., 6723 Bertner Dr., Houston.
Rosenbalm, Thomas J.,
990 N. Federal Rd., Houston.
Rosenberg, Harvey S., 6621 Fannin, Houston.
Rosenthal, Morris Wm.,
8561 Long Point Rd., Houston.
Rosenthal, Paul G., (Int.)
3014 Shenandoah, Houston.
Rothenberg, Lionel H., 618 Tatar St., Pasadena.
Roy, Harry L., Jr.,
8561 Long Point Rd., Houston.
Royce, Thomas L.,
Hermann Prof. Bldg., Houston.
Ruiz, John J., 7919 Park Pl. Blvd., Houston.
Ruiz, Richard S., (Int.),
Kresge Eye Inst., Detroit, Mich.
Rumph, Quah, 821 Med. Arts Bldg., Houston.
Russell, Thomas G.,
5000 Montrose Blvd., Houston.
Russell, William O.,
6723 Bertner Blvd., Houston.
Rutledge, Felix N., 4828 Caroline, Houston.
Ryan, Andrew J., (Int.),
Hermann Hosp., Houston.
Ryan, Bert M.,
1015 E. Thomas Ave., Pasadena.
Ryan, James C., 1516 Pierce Ave., Houston.
Ryan, James E., 2103 DeMilo Dr., Houston.
Sacco, Allan C., 1102 Elgin, Houston.
Saied, Victor C., (Int.), V. A. Hosp., Houston.
Salerno, Joseph P., 1717 La Branch, Houston.
Salinger, Alfons, 2212 Calumet Dr., Houston.
Salmeron, Pedro, Jr., 2409 Canal St., Houston.
Salmon, George W.,
3911 Montrose Blvd., Houston.
Salyer, Charles P., 3004 Little York, Houston.
Sammons, James H.,
111 San Jacinto St., Highlands.
Sammons, Karl T., Box 684, Highlands.
Samuels, Melvin L.,
M. D. Anderson Hosp., Houston.
Sanders, Charles B.,
1526 Med. Arts Bldg., Houston.
Sanders, Elmer K.,
Hermann Prof. Bldg., Houston.
Sanders, Zal H., 1318 Telephone Rd., Houston.
Sanderson, Thomas A.,
4974 Valkeith, Houston.
Sappington, H. O., Box 4128, Baytown.
Scardino, Peter H., (Hon.),
4018 Meadow Lake, Houston.
Schafer, Ted A.,
6243 Old Richmond Rd., Houston.
Schaffer, Elliott N., 1208 Wayside, Houston.
Schaffer, Helen G.,
8525 Stella Link Rd., Houston.
Schaffer, Randolph L.,
102 Med. Towers, Houston.
Schaffer, Samuel S.,
8525 Stella Link Rd., Houston.
Schaffner, Harold B.,
3102 San Jacinto, Houston.
Scheid, Martin F., (Int.),
Hermann Hosp., Houston.
Scher, David, 814 Med. Towers, Houston.
Scheurich, Richard E.,
105 E. Texas Ave., Baytown.

Schiflett, J. Ray, II, Box 45875, Houston.
Schmitt, John R., 4528 Griggs Rd., Houston.
Schnur, Roxana R., 5127 Jackwood, Bellaire.
Schnur, Sidney, Med. Arts Bldg., Houston.
Schoenvogel, Otto F., Jr.,
Hermann Prof. Bldg., Houston.
Schofield, James Roy,
1200 M. D. Anderson Blvd., Houston.
Schrum, David I., 2229 Dorrington, Houston.
Schuhmacher, L. F., Jr.,
Hermann Hospital, Houston.
Schuleman, Israel H.,
918 Med. Towers, Houston.
Schultz, Herman J.,
806 Hermann Prof. Bldg., Houston.
Schultz, Jacob F.,
1616 Mem. Prof. Bldg., Houston.
Schwartz, Harold R.,
1215 Med. Towers, Houston.
Schwartz, Max J., 1204 Wayside Ave., Houston.
Scott, Dan W., 1818 Crawford St., Houston.
Scott, F. Brantley, Jr., (Int.),
3744 Jardin, Houston.
Scott, John H., Box 3805, Baytown.
Scott, Russell, Jr.,
1200 M. D. Anderson Blvd., Houston.
Scott, Warren M., Jr., (Int.),
V. A. Hospital, Houston.
Seale, Everett R.,
1217 Hermann Prof. Bldg., Houston.
Searle, Noel B., (Int.),
4130 Turnberry Cir., Houston.
Sears, Ernest S., 346 W. 19th St., Houston.
Sears, Mary E.,
M. D. Anderson Hosp., Houston.
Selders, Raymond E.,
1601 S. Shepherd, Houston.
Selke, Oscar O., Jr.,
615 Hermann Prof. Bldg., Houston.
Sellers, Dunham R.,
112 Meyerland Plaza Mall, Houston.
Sengelmann, W. A.,
1423 Mem. Prof. Bldg., Houston.
Sens, Franklin E., (Int.),
Hermann Hospital, Houston.
Seybold, Herbert M., 5420 Caroline, Houston.
Seybold, William D.,
Hermann Prof. Bldg., Houston.
Shapiro, Edward M., 624 Tatar St., Pasadena.
Sharkey, Paul C.,
1023 Lyndon, Apt. 12, Houston.
Sharp, William E., 2800 Garth Rd., Baytown.
Shaw, Edward N., 1401 Eagle Ave., Houston.
Shaw, Leroy B., 8363 Winkler Dr., Houston.
Shearer, Thomas P.,
Hermann Prof. Bldg., Houston.
Shelton, Elvin L., Jr.,
Hermann Prof. Bldg., Houston.
Sher, Benjamin, 421 Pinehaven, Houston.
Sher, Malcolm F.,
515 Hermann Prof. Bldg., Houston.
Sherrill, Douglas B., 617 Tatar St., Pasadena.
Sherrill, E. Allen, Jr.,
3618 Mt. Vernon, Houston.
Sherrill, William M.,
3618 Mt. Vernon, Houston.
Shields, John C., 624 Tatar St., Pasadena.
Shindler, Thomas O.,
Hermann Prof. Bldg., Houston.
Shoemaker, Emitt H.,
5000 Montrose Blvd., Houston.
Shoemaker, Roger D.,
410 West 20th St., Houston.
Short, Dennis W.,
Hermann Prof. Bldg., Houston.
Shoss, Joseph,
10521 S. Post Oak Rd., Houston.
Shrader, Harold E.,
906 E. Southmore St., Pasadena.
Shullenberger, C. C.,
6723 Bertner Blvd., Houston.
Shutts, Paul E., 5326 Calhoun Rd., Houston.
Siegel, Jacob, 7410 Long Point Rd., Houston.
Silverblatt, Julian,
901 Hermann Prof. Bldg., Houston.
Silverman, Robert A., 1603 Lorraine, Houston.
Simkin, Samuel, 1606 Med. Towers, Houston.
Sinclair, Marga H., 703 Med. Towers, Houston.
Sinclair, Thomas A.,
333 W. 20th St., Houston.
Singleton, Edward B., 6621 Fannin, Houston.
Singleton, Paul C. A.,
6410 Fannin St., Houston.
Skaggs, Ray H., Hermann Prof. Bldg., Houston.
Skelton, James M., 2708 Weslayan, Houston.
Skinner, Samuel J.,
Hermann Hospital, Houston.
Skogland, John E., 3200 Caroline St., Houston.
Slataper, Eugene L., The Med. Towers, Houston.
Slaughter, Carlos A., Box 217, Sugarland.
Smith, Alfred E., 1746 Ebony, Houston.
Smith, Burt B., 5101 Caroline, Houston.
Smith, Clifford T., (Inac.),
3461 Piping Rock, Houston.
Smith, Edward T., 6410 Fannin St., Houston.
Smith, Harry S., 2708 Weslayan St., Houston.
Smith, J. Murry, Med. Arts Bldg., Houston.
Smith, J. Leslie, Jr.,
M. D. Anderson Hosp., Houston.
Smith, P. Woodbury,
2255 W. Holcombe, Houston.
Smith, Thomas E., 3520 Market St., Baytown.
Smolens, Nathan M., (Mil.),
U. S. A. H., Fort Hood.
Snow, William J.,
708 Med. Arts Bldg., Houston.
Snyder, Hal, 4520 Rossmoyne, Houston.
Snyder, Harvey B., 1216 Main St., Houston.
Snyder, John T., Box 708, Channelview.
Sonenthal, I. Ronald,
2219 Dorrington, Houston.
Sosebee-Crump, Winnie J.,
601 Med. Arts Bldg., Houston.
Spain, Charles A., (Int.),
6722 Sharpcrest Lane, Houston.
Spankus, Willerd H., 3527 Ella Blvd., Houston.
Speck, Carlos D., Jr.,
3709 Montrose Blvd., Houston.
Speidel, Thomas H.,
1010 N. Federal Rd., Houston.
Spence, Philip S., Jr.,
1037 Esperson Bldg., Houston.
Spencer, William A., Box 20095, Houston.
Spezia, Joseph L., 4832 Caroline, Houston.
Spiller, Rachel F.,
5000 Montrose Blvd., Houston.
Spiller, William F., Jr.,
5000 Montrose Blvd., Houston.
Sponsel, Thane T.,
1002 Hermann Prof. Bldg., Houston.
Stackhouse, Howard, Jr.,
1222 Med. Arts Bldg., Houston.
Stahl, David M., 1504 Med. Towers, Houston.
Stalnaker, Paul R., (Hon.),
4715 Fannin, Houston.
Stanford, John P., Jr.,
1409 Mem. Prof. Bldg., Houston.
Staub, John D., 2708 Weslayan, Houston.
Stehlin, John S., Jr.,
6723 Bertner Dr., Houston.
Stent, Paul A.,
3501 Oak Forest Dr., Houston.
Stephens, Jerry H., 3614 Albans Rd., Houston.
Stephens, Mollie M.,
1025 S. Wafer, Pasadena.
Stephenson, Betty P.,
6516 Bertner Ave., Houston.
Stephenson, Charles T.,
10 Chelsea Place, Houston.
Sterling, Russell R., (dead), Houston.
Sterns, Jerome E., 1114 Med. Towers, Houston.
Stevenson, Murphy D.,
Hermann Prof. Bldg., Houston.
Stone, John S., 4223 Lyons Ave., Houston.
Stool, Joseph A., 2417 Glen Haven, Houston.
Storey, Sharon S., Jr.,
6534 Wynnwood St., Houston.
Stork, Walter J., 816 Med. Towers, Houston.
Stough, John T., 4501 Fannin St., Houston.
Strashun, Mat F., 1811 Crawford St., Houston.
Strassmann, Erwin O.,
Hermann Prof. Bldg., Houston.
Straw, Kenneth, 3507 Reveille, Houston.
Stuermer, Cassie E.,
13131 Rockglen Dr., Houston.
Subotnik, Nathan,
6425 Richmond Rd., Houston.
Sullivan, Margaret P.,
M. D. Anderson Hosp., Houston.
Surgi, Marie L., 3709 Montrose Blvd., Houston.
Sutow, Wataru W.,
M. D. Anderson Hosp., Houston.
Sutton, William C., (Int.),
Hermann Hospital, Houston.
Synnott, James D., 333 W. 20th St., Houston.
Synnott, Thomas G., 410 W. 20th St., Houston.
Talley, Arthur T., Jr.,
1307 Med. Arts Bldg., Houston.
Talley, Elizabeth D.,
11757 Taylor Crest, Houston.
Talty, Matthew H., Jr.,
1809 Crawford, Houston.
Tashnek, Arthur B.,
1411 Med. Towers, Houston.
Tatar, Mervin, 617 Tatar St., Pasadena.
Tausend, Harold J., 5012 Travis St., Houston.
Tausend, Paul L., 1029 E. Thomas, Pasadena.
Taylor, Fred M.,
Baylor Med. College, Houston.
Taylor, H. Newell, No. 2 Chelsea Pl., Houston.
Taylor, Harvey Grant,
M. D. Anderson Hosp., Houston.
Taylor, Holman, Jr.,
Hermann Prof. Bldg., Houston.
Taylor, Homer A., Jr.,
1406 Mem. Prof. Bldg., Houston.
Telford, Jane, 2010 Banks, Houston.
Teng, Ching Tseng, 6621 Fannin, Houston.
Tengg, Nicholas E., 3842 Glen Arbor, Houston.
Tharp, Warren B., 3921 Richmond, Houston.
Thoma, Earl W., Med. Arts Bldg., Houston.
Thomas, Albert M.,
938 Hermann Prof. Bldg., Houston.
Thomas, Fred P.,
Hermann Hospital, Houston.
Thomas, George B.,
5000 Montrose Blvd., Houston.
Thomas, John R., 9402 Cadman Ct., Houston.
Thomas, Sellers J., Jr.,
508 Hermann Prof. Bldg., Houston.
Thomas, Stephen, 4040 Broadway, Houston.
Thomason, Edgar M., 2136 Yale St., Houston.
Thompson, B. D., 5655 Briar Dr., Houston.
Thompson, Raymond D.,
8346 Long Point Rd., Houston.
Thompson, Walker, 4610 Cedar Oaks, Bellaire.
Thorn, Sylvester W., 211 Kincaid Dr., Houston.
Thorning, W. B., Jr.,
4119 Montrose Blvd., Houston.
Tindel, Roland L.,
846 Oak Valley Dr., Houston.
Tips, Eugene F., 802 Med. Towers, Houston.
Topek, Nathan H.,
1426 Med. Arts Bldg., Houston.
Torno, Curtis J., 1029 Thomas St., Pasadena.
Trible, John B.,
1625 Med. Arts Bldg., Houston.
Tucker, Eli J., 610 Med. Arts Bldg., Houston.
Tucker, J. Norris,
526 Med. Arts Bldg., Houston.
Tucker, Sheldon M., 104 E. 20th St., Houston.
Turboff, Sidney W., 1603 Lorraine, Houston.
Turner, B. Weems, (Hon.),
1700 Holcombe Blvd., Houston.
Turner, Louis E.,
601 Med. Arts Bldg., Houston.
Turner, Russell L., Jr., 2136 Yale, Houston.
Tusa, Theo S., Esperson Bldg., Houston.
Tusa, Theo S., Jr.,
1608 Med. Towers, Houston.
Tuttle, L. L. D.,
Hermann Prof. Bldg., Houston.
Tyner, Furman H.,
704 Med. Arts Bldg., Houston.
Ulert, Izaak A.,
8525 Stella Link Rd., Houston.
Upchurch, Kent P., 618 Tatar St., Pasadena.
Usher, Francis C.,
Hermann Prof. Bldg., Houston.
VandenBossche, L. J.,
104 East 20th St., Houston.
Vandivier, Thomas G.,
6448 Fannin St., Houston.
Van Schoote, A. M., (Int.),
6723 Bertner Dr., Houston.
Van Velzer, David A.,
3415 Bellaire Blvd., Houston.
Vanzant, Thomas J.,
Hermann Prof. Bldg., Houston.
Vaughan, Luther M.,
Hermann Hospital, Houston.
Veatch, Everett P., 211 S. Main, Pasadena.
Vick, J. Louise, 4115 Caroline, Houston.
Viehweg, Wayne R.,
1528 Med. Arts Bldg., Houston.
Vincent, Terry S., 2012 Crawford, Houston.
Vine, Bernard G., (Int.),
5406 Schumacher Lane, Houston.
Vint, William A., 1004 Seymour, Pasadena.
Vlahakos, Antonio B., 211 S. Main, Pasadena.
Von Pohle, Kenneth C.,
404 Hermann Prof. Bldg., Houston.
Wachsman, David V.,
1319 Austin St., Houston.
Wagner, E. Lionel,
4109 Montrose Blvd., Houston.
Wagner, Robert G., 2039 Dryden Rd., Houston.
Wahlen, Henry E.,
5428 Northwood Dr., Baltimore, Md.
Waldron, George W.,
600 Hermann Prof. Bldg., Houston.
Walker, Jack L., 105 N. Rice Ave., Bellaire.
Walker, Joseph D., 520 W. 20th St., Houston.
Walker, Rhey, II,
4705 Montrose Blvd., Houston.
Wall, Herman A., Jr.,
106 W. Jackson St., Pasadena.
Wall, John A., Hermann Prof. Bldg., Houston.
Wallace, Grover C.,
906 E. Southmore St., Pasadena.
Wallace, Stuart A.,
1200 M. D. Anderson Blvd., Houston.
Waller, Jester J., Jr., 104 E. 20th St., Houston.
Wallis, Marshall, (Hon.), Rt. 1, Harlingen.
Wallis, William M., 218 Main St., Houston.
Walmsley, George L., 721 E. Texas, Baytown.
Walpole, Ben, 3101 Fannin St., Houston.
Walter, Paul J., Hermann Prof. Bldg., Houston.
Walton, Ida K., 719 E. Texas Ave., Baytown.
Walton, Worth, 721 E. Texas Ave., Baytown.
Warner, Clyde M., (Inac.),
3256 Reba St., Houston.
Warner, Lucien M., (Inac.),
1521 Bonnie Brae, Houston.

Warren, Joseph F., Tomball.
Waters, Charles R., Box 505, Highlands.
Watson, James E., Jr.,
Hermann Prof. Bldg., Houston.
Watson, Thomas D., Jr.,
2071 Brentwood Dr., Houston.
Webb, Edward D., 5608 Yale, Houston.
Weeden, Kenneth C.,
3527 Ella Blvd., Houston.
Weil, Sol B., Jr., 2246 Dryden Rd., Houston.
Welch, Beatrice,
5124 Old Richmond Rd., Bellaire.
Welch, Jay J.,
1210 Mem. Prof. Bldg., Houston.
Wells, Benjamin T., (Mil.),
1208 6th Ave., S.E., Rochester, Minn.
Welsh, Hugh C., 2715 Fannin, Houston.
West, Joseph E., Jr., 7338 McHenry, Houston.
Westbrook, Orian C.,
1207 Hermann Prof. Bldg., Houston.
Westmoreland, Jack C.,
4562 Old Richmond Rd., Bellaire.
Westover, Harry M., (Hon.),
8409 Jensen Dr., Houston.
Wexler, Alvin S.,
6303 S. Park Blvd., Houston.
Wheeler, Clarence J., Jr.,
940 Hermann Prof. Bldg., Houston.
Wheeless, John V., Jr.,
1408 Hermann Prof. Bldg., Houston.
White, Adair W., Jr.,
1820 W. 43rd St., Houston.
White, Ben B., 411 W. 20th St., Houston.
White, Edgar C.,
6723 Bertner Blvd., Houston.
White, John H., 618 S. Tatar St., Pasadena.
Whitehurst, James R.,
Hermann Prof. Bldg., Houston.
Whitsitt, James J.,
Mellie Esperson Bldg., Houston.
Whitson, Robert O.,
1003 Polly Ave., Baytown.
Wible, D. Jordan, 3304 Milam, Houston.
Wiesenthal, Joseph, 200 Wayside, Houston.
Wigby, Palmer E.,
Hermann Prof. Bldg., Houston.
Wilber, Stewart A.,
M. D. Anderson Hosp., Houston.
Wilbourn, George R.,
904 E. Southmore, Pasadena.
Wilkerson, Bernie J.,
1417 Mem. Prof. Bldg., Houston.
Wilkerson, Edward A.,
910 Hermann Prof. Bldg., Houston.
Wilkin, Mabel G.,
1400 Hermann Dr., Houston.
Williams, Allen G., 624 S. Tatar, Pasadena.
Williams, Bryan V.,
1704 Crawford St., Houston.
Williams, E. Carson,
1017 Hermann Prof. Bldg., Houston.
Williams, Philip D., Jr.,
2701 Berry Rd., Houston.
Williamson, Warren J.,
2404 Rice Blvd., Houston.
Williamson, Willie A.,
3804 Broadway, Houston.
Wills, Seward H.,
Hermann Prof. Bldg., Houston.
Wilson, Anthony,
906 E. Southmore, Pasadena.
Wilson, Arnold,
906 E. Southmore, Pasadena.
Wilson, Carl S., 1512 W. Alabama, Houston.
Wilson, R. T., (Hon.),
12601 Memorial Dr., Houston.
Wilson, R. Bruce,
1000 N. Federal Rd., Houston.
Wilson, Roy D., (Mil.),
514 Ferson Loop, San Antonio.
Windrow, Frank M.,
Hermann Prof. Bldg., Houston.
Windrow, Nuel C., Jr.,
2405 Tangley St., Houston.
Wing, Herman,
1200 M. D. Anderson Blvd., Houston.
Winograd, Eugene,
1708 Houston Blvd., South Houston.
Wise, Robert A., P. O. Box 2180, Houston.
Wise, Robert J.,
Hermann Prof. Bldg., Houston.
Wise, Samuel P., III,
5807 Warm Springs Rd., Houston.
Withers, Ben T.,
Hermann Prof. Bldg., Houston.
Withers, Henry W., 3102 San Jacinto, Houston.
Wolever, Thomas H. S.,
1200 M. D. Anderson Blvd., Houston.
Wolf, Charles L., 1818 Caroline St., Houston.
Wolf, E. Trowbridge,
4411 Fannin St., Houston.
Wolf, Walter J., 1402 Med. Towers, Houston.
Wolfe, Russell S., 5101 Almeda, Houston.
Wolters, Carlton E., 5503 Crawford, Houston.
Wood, Harold,
230 Med. Arts Bldg., Houston.
Wood, Harold A., (Mil.), 705 Baca, Houston.
Woolf, Martin P., 907 S. Tatar, Pasadena.
Wootters, John H.,
Hermann Prof. Bldg., Houston.
Wortham, G. Forrest, Jr.,
2708 Weslayan St., Houston.
Wright, Ernest, 2407 N. Main St., Houston.
Wright, William K.,
Hermann Prof. Bldg., Houston.
Wyatt, Malcolm H., 114 N. Second, LaPorte.
Yaege, Arlo O., Box 728, Tomball.
Yates, Charles W., 2611 Ave. G, Rosenberg.
Yates, William H.,
10923 Chimney Rock, Houston.
Yoder, Nelson J., 720 W. Main St., LaPorte.
Yollick, Bernard L.,
The Medical Towers, Houston.
York, Byron P.,
3911 Montrose Blvd., Houston.
Young, Carl B.,
Continental Bank Bldg., Houston.
Young, David B., 106 W. Jackson, Pasadena.
Young, John B., 3837 Bellaire Blvd., Houston.
Young, Ozora F., 6026 Milart St., Houston.
Youngblood, Jarvis C.,
3011 Ella Lee Lane, Houston.
Younkin, Charles R.,
6243 Richmond Rd., Houston.
Yow, Ellard M.,
Baylor U. College of Med., Houston.
Zanek, Otto L., 1610 Med. Towers, Houston.
Zax, Emile,
515 Hermann Prof. Bldg., Houston.
Zeis, Leander B.,
354 Hermann Prof. Bldg., Houston.
Zeluff, George W.,
Hermann Prof. Bldg., Houston.
Zepeda, Rudolph F., 4030 Roseneath, Houston.
Zimmerman, Stanley J., (Int.),
Hermann Hospital, Houston.
Zionts, Martin A., 3507 Louisiana, Houston.

MONTGOMERY

Bell, Henry G., 101 E. Phillips St., Conroe.
Brignac, Louis F., Jr., 316½ W. Davis, Conroe.
Carroll, I. Emil, Conroe.
Holland, William M., Conroe.
Hurst, William R., Ave. G & Crooke, Conroe.
Hutchins, Melvin G., 324½ N. Main, Conroe.
Lenon, EE., Conroe.
Powell, Burrell E., Powell Clinic, Conroe.
Sadler, Deane L., Conroe.
Watson, Irving M., Jr., Conroe.
Wilkerson, Walter D., Jr.,
Ave. G & Crooke, Conroe.
Wilkins, Afton N., 213 S. 1st St., Conroe.

POLK-SAN JACINTO

Corso, John, Livingston.
Dabney, Joseph T., Jr., Livingston.
Dameron, James H., Livingston.
Gardner, Thomas L., Livingston.
Hooks, Murray D., Corrigan.
Mitchell, L. Du-Wayne,
P. O. Box 51, Shepherd.

WALKER-MADISON-TRINITY

Addison, Eugene M., 1429 13th St., Huntsville.
Autrey, Stacy L., Trinity.
Barnes, Sam R., Trinity.
Black, F. Ray, 1312 11th St., Huntsville.
Blalock, Raymond B., Huntsville.
Boriack, Leroy A.,
1520 W. 11th St., Huntsville.
Bush, Leonard E., Box 328, Huntsville.
Cole, Thomas C., 1203 Ave. J, Huntsville.
Cook, John R., Box 32, Trinity.
Goodrich, William A., Sr., Huntsville.
Hanson, Minter D., Huntsville.
Heath, Jesse B., Madisonville.
Thornton, Harold H., Trinity.
Veazey, William B., 1203 Ave. J, Huntsville.
Woodward, W. Mac, Huntsville.

WASHINGTON-BURLESON

Dodd, Tilman E., 2710 Enfield Rd., Austin.
Gallo, Albert E., 204 S. Echols, Caldwell.
Giddings, Thomas H., 604 Stone St., Brenham.
Hasskarl, Robert A., Box 696, Brenham.
Hasskarl, Walter F., Sr.,
203 E. Academy, Brenham.
Hasskarl, Walter F., Jr.,
203 E. Academy, Brenham.
Hodde, Fred H., Box 601, Brenham.
Hodde, Herman O., 208 W. Main, Brenham.
Johnson, Joel H., 509 E. Academy, Brenham.
Knolle, Roger E., 106½ E. Alamo, Brenham.
Knolle, Waldo A., (dead), Brenham.
Owen, A. George, (Inac.), Rt. 5, Brenham.
Pazdral, George V., Somerville.
Schoenvogel, Clarence W.,
Schoenvogel Clinic, Brenham.
Schoenvogel, Otto F.,
Schoenvogel Clinic, Brenham.
Schoenvogel, Robert L.,
Citizens Phar Bldg., Brenham.
Siptak, John E., Caldwell.
Smith, Joe C., Caldwell.
Southern, Charles E.,
Southern Clin. Bldg., Brenham.
Steinbach, Herbert L.,
Med. Arts Bldg., Brenham.
Tottenham, Edwin P.,
106 W. Vulcan St., Brenham.

TENTH DISTRICT

Dr. Stephen B. Tucker, Nacogdoches, Councilor

ANGELINA

Allen, Cedric A., 442 Lake St., Lufkin.
Arnett, Robert K., 302 N. Raguet, Lufkin.
Atkinson, Basil E., Jr., 923 Ellis Ave., Lufkin.
Barrett, John G., 1109 Ellis Ave., Lufkin.
Brashear, Doyle H., (Int.),
Station A, Salem, Ore.
Bryan, Arthur F., Box 107, Camden.
Clement, J. Carroll, 110 S. Raguet, Lufkin.
Clements, Peyton C., 402 S. First, Lufkin.
Connell, Anna Beth, 1000 Frank Ave., Lufkin.
Denman, Byford H., 221 S. First St., Lufkin.
Denman, Linwood H., Box 354, Lufkin.
Denman, Peyton L., 302 S. Raguet, Lufkin.
Evans, Charles W., Jr., 1022 Ellis Ave., Lufkin.
Hollingsworth, H. W.,
5105 Neptune Way, Tampa, Fla.
Hoot, Edwin L., Box 307, Diboll.
Hyman, Bernard, Box 176, Lufkin.
Klein, James C., Box 1216, Lufkin.
Koch, John C., 1306 Frank Ave., Lufkin.
Laughlin, John Milton, 919 Ellis Ave., Lufkin.
Martin, Juan E., 923 Ellis, Lufkin.
McVicker, Bobby H., Box 1076, Lufkin.
Medford, U. Gail, 110 S. Raguet St., Lufkin.
Mitchell, William A.,
201 Hemenway Bldg., Lufkin.
Orman, Forrest C., 1111 Ellis Ave., Lufkin.
Peebles, Felix, Jr., 419 Perry Bldg., Lufkin.
Pruitt, George Jack, Box 133, Lufkin.
Read, Royce E., 818 Frank Ave., Lufkin.
Seitz, Ernest R., 302 N. Raguet St., Lufkin.
Shepard, Groom S., 1121 Ellis Ave., Lufkin.
Spivey, Dan, 818 Frank, Lufkin.
Taylor, Robert W., 118½ S. First, Lufkin.
Thames, William D., Jr.,
818 Frank Ave., Lufkin.
Thannisch, George E., 1121 Ellis Ave., Lufkin.
Tinkle, Lassiter T., 1608 S. First St., Lufkin.
Wade, Jack H., Box 1300, Lufkin.

HARDIN-TYLER

Burton, L. Gayle, Box 788, Woodville.
Copeland, Sam P., Jr., 735 N. Fifth, Silsbee.
Gaunt, Eugenia T., Kountze.
Gilchrist, John Q., Woodville.
Hooks, Henry A., Kountze.
Parker, Wade M., 735 N. 5th, Silsbee.
Poshataske, Wallace J.,
510 N. Fifth St., Silsbee.
Ryan, Robert L., 821 Barkley St., Sour Lake.
Spidle, James L., Box 446, Sour Lake.
Swearingen, Robt. E., Box 608, Woodville.
Tate, John H., Kountze Clinic, Kountze.
Tate, Robert A., (Inac.), Kountze.
Tennison, George D.,
445 Roosevelt Dr., Silsbee.

JASPER-NEWTON

Dickerson, Joe W., Jasper.
Jones, Thomas R., 139 W. Lamar, Jasper.
Lewis, Theodore C., Jr.,
Hardy-Hancock Hosp., Jasper.
Love, Albert J., Newton.
Mattingly, Claude, Jr.,
254 N. Wheeler St., Jasper.
McCreight, William F., (Hon.),
P. O. Box 218, Kirbyville.
McGrath, J. J., Jasper.
Moore, John T., Box 638, Kirbyville.
Neubauer, Dorothea E., Box 206, Pineland.
Richardson, A. J., Jr., Jasper.
Stockdale, Rider E.,
Hardy Hancock Hosp., Jasper.
Winslow, Grover C., Hemphill Clin., Hemphill.

JEFFERSON

Adams, Barnette E. J., Jr.,
1950 Ninth Ave., Port Arthur.
Adams, Robert M., P. O. Box 878, Groves.
Adkins, Charles F., 675 Fifth St., Beaumont.
Alexander, Hugh E.,
2372 Calder Ave., Beaumont.
Allamon, Emmett L.,
918 Goodhue Bldg., Beaumont.
Allison, F. Peel, Hotel Beaumont, Beaumont.
Almaraz, Herman,
2749 Procter St., Port Arthur.
Anderson, Roland B.,
1950 Ninth Ave., Port Arthur.
Bailey, Percy W., Jr.,
675 Fifth St., Beaumont.
Barr, H. Buford, 501 Perlstein Bldg., Beaumont.
Bebeau, Eugene R., 3495 Crestwood, Beaumont.
Beck, Edmund J., 1110 Longfellow, Beaumont.
Bell, Walter L., Box 288, Port Arthur.
Bevil, H. Grady, 301 Doctors Bldg., Beaumont.
Bevil, Harold H.,
1110 Longfellow Dr., Beaumont.
Bevil, John R., (Hon.),
2625 Laurel Ave., Beaumont.
Bevil, Lamar C., 2625 Laurel Ave., Beaumont.
Beyt, Frank J., 235 Adams Bldg., Port Arthur.
Blackwood, James Q.,
701 Goodhue Bldg., Beaumont.
Blum, Sigmund L., 573 Pearl St., Beaumont.
Bourgeois, Richard J.,
2301 Procter St., Port Arthur.
Boyle, Frank B., 2248 Procter St., Port Arthur.
Brandau, William H.,
703 Amer. Natl. Bank Bldg., Beaumont.
Bridges, James L.,
2190 Harrison St., Beaumont.
Brittain, Robert D., Jr.,
2372 Calder Ave., Beaumont.
Brown, Walter D., (Hon.),
2262 North St., Beaumont.
Browne, Robert S., Adams Bldg., Port Arthur.
Brownrigg, Thos. H.,
2540 Broadway, Beaumont.
Bruce, Charles O., Box 817, Port Neches.
Bybee, Joe A., 400 Center St., Beaumont.
Byram, Dan H., 2600 Procter St., Port Arthur.
Byrd, Houston F., 1322 Magnolia, Port Neches.
Byrd, Lee Roy, Jr., Box 701, Port Arthur.
Byrd, Peter G., 695½ Forsythe, Beaumont.
Caldwell, Pearson C.,
506 Goodhue Bldg., Beaumont.
Campbell, George L.,
3155 Stagg Dr., Beaumont.
Carabelle, R. Wm., (Int.),
Ark. Baptist Hosp., Little Rock, Ark.
Carter, John H.,
460 First Fed. Sav. Bldg., Beaumont.
Carter, Louian C.,
2600 Procter St., Port Arthur.
Caskey, Charles J.,
1785 Washington, Beaumont.
Catalano, Russell J.,
502 Goodhue Bldg., Beaumont.
Chapman, Frank C.,
5650 Azalea Dr., Beaumont.
Chevis, Edward G.,
525 Ninth Ave., Port Arthur.
Chiasson, John L., (Hon.),
1118 Dearing St., Port Neches.
Chiasson, Mary P.,
261 E. Circuit Dr., Beaumont.
Christie, Alonzo B., Jr.,
Noakes St., Corpus Christi.
Cochran, E. Winston, 675 Fifth St., Beaumont.
Colby, Fred W. C., 2380 Hazel St., Beaumont.
Coleman, Charles W.,
540 Center St., Beaumont.
Colletti, Ben M., 601 Ninth Ave., Port Arthur.
Couch, Ray E., Box 3277, Port Arthur.
Crager, Jay C., 601 Goodhue Bldg., Beaumont.
Crawford, Geo. F., 3155 Stagg Dr., Beaumont.
Curry, Dwight E., (Inac.),
2600 Procter St., Port Arthur.
Darnell, B. C., 4620 Gladys St., Beaumont.
Derrick, Joseph P.,
2190 Harrison St., Beaumont.
Douglas, Jake S., 935 Buford St., Beaumont.
Drawhorn, Chester W.,
917 Port Neches Ave., Port Neches.
Duplan, Don C., Jr.,
4627 Alamosa Dr., Port Arthur.
Duren, Norman, 3155 Stagg Dr., Beaumont.
Durso, Joseph, 949 7th St., Port Arthur.
Echols, Obie L., 634 Ave. C, Port Neches.
Eisenstadt, Heinz B.,
2301 Procter St., Port Arthur.
Elster, Benjamin B.,
2301 Procter St., Port Arthur.
English, Dudley M., 550 Tenth St., Beaumont.
Erhard, Peter S., Box 294, Beaumont.
Esslinger, Josh J., 301 Doctors Bldg., Beaumont.
Everett, Eva T., 1950 Ninth Ave., Port Arthur.
Falgout, Frank J., Winnie.
Fama, Joseph R., 3360 Fannin, Beaumont.
Faulk, M. Edmund, Jr.,
675 Fifth St., Beaumont.
Fears, T. Alvin, Goodhue Bldg., Beaumont.
Ferguson, Edward C.,
Goodhue Bldg., Beaumont.
Fertitta, Julian J., 590 Center St., Beaumont.
Fertitta, Sam J., Jr., 570 Center St., Beaumont.
Fortney, Paul N., 550 Tenth St., Beaumont.
Fulbright, Carl W., (Inac.),
4230 Sunken Cts., Port Arthur.
Fuqua, Wm. Cruse, 3155 Stagg Dr., Beaumont.
Furey, Ellen D., 1025 Ave. B, Beaumont.
Fuselier, Julius D., 749 16th St., Port Arthur.
Gammill, Ralph R.,
415 Goodhue Bldg., Beaumont.
Gardner, John N.,
613 Goodhue Bldg., Beaumont.
Giblin, Hillard J., 6285 39th St., Groves.
Gibson, U. G., 1000 Texas Ave., Port Arthur.
Gill, George G., 370 Tenth St., Beaumont.
Gish, James R., 3155 Stagg Dr., Beaumont.
Glass, Cecil R., 2462 Calder Ave., Beaumont.
Glass, Walter W., Jr.,
2130 Memorial Blvd., Port Arthur.
Goldblum, Harvey H.,
3048 Procter St., Port Arthur.
Graber, W. J.,
610 Amer. Natl. Bank Bldg., Beaumont.
Greenberg, Philip B.,
502 Perlstein Bldg., Beaumont.
Greene, W. Leonard, 670 7th St., Beaumont.
Hager, Dale C., 2040 Park St., Beaumont.
Haig, Martin R.,
2130 Memorial Blvd., Port Arthur.
Hamill, James E., (Mil.),
2301 Procter St., Port Arthur.
Harlan, H. D., (Inac.),
San Jacinto Bldg., Beaumont.
Harper, J. Y., 849 Fifth St., Port Arthur.
Hart, Frank B., 713 Mariposa St., Beaumont.
Hart, John A., 3280 Fannin St., Beaumont.
Hartel, Charles J., 2544 Broadway, Beaumont.
Hartman, Lee E., (Int.),
2612 Norfolk, Houston.
Hartman, Sam F., 1292 Liberty Ave., Beaumont.
Healey, Gordon, 535 Stadium Rd., Port Arthur.
Heare, Charles R., 390 Tenth St., Beaumont.
Heare, Louis C., Box 312, Port Arthur.
Henderson, John A.,
3155 Stagg Dr., Beaumont.
Hennington, H. M.,
723 Washington Blvd., Beaumont.
Hill, Isaac H., 901 Fifth St., Port Arthur.
Hille, Charles D.,
1535 Martin Dr., Port Arthur.
Hinshaw, Arvil J.,
3155 Stagg Dr., Beaumont.
Hitt, Rankin V., Jr.,
3155 Stagg Dr., Beaumont.
Holman, Winston C.,
1931 Ninth Ave., Port Arthur.
Holmes, Glen I.,
Stagg Dr. & 8th St., Beaumont.
Hosen, Harris, 2649 Procter St., Port Arthur.
Hyman, Barnett M.,
First Fed. Sav. Bldg., Beaumont.
Ivers, James B., 3155 Stagg Dr., Beaumont.
Jack, Laurine D., 470 N. Freeway, Beaumont.
Jacobson, Harry,
523 Perlstein Bldg., Beaumont.
Johnson, Jerry, 3155 Stagg Dr., Beaumont.
Jones, E. Harrold, 2625 Laurel Ave., Beaumont.
Kalbaugh, Elmore P., Jr.,
1950 Ninth Ave., Port Arthur.
Keith, Frank R., P. O. Box 110, Port Arthur.
Kennedy, Jeretta I., P. O. Box 43, Nome.
Kern, Paul J., 1104 Port Neches, Port Neches.
Killingsworth, W. Price,
541 9th Ave., Port Arthur.
Knight, Max J., 900 9th Ave., Port Arthur.
Koshkin, Bernard D., 3360 Fannin, Beaumont.
Kuhlman, Fred Y.,
1950 Ninth Ave., Port Arthur.
LaCour, Edward G., 3155 Stagg Dr., Beaumont.
Lancaster, Lifford R.,
303 Doctors Bldg., Beaumont.
Lanier, Ernest W., 6272 Jefferson St., Groves.
Larkins, L. Curtis, 755 College St., Beaumont.
Ledbetter, L. H., 550 10th St., Beaumont.
Lewis, James W., 2649 Procter St., Port Arthur.
Lewis, Seab J., 2620 Laurel Ave., Beaumont.
Lindsey, Eugene H.,
2372 Calder Ave., Beaumont.
Loewenstein, Joseph M.,
3048 Procter St., Port Arthur.
Lombardo, R. T., 810 19th St., Beaumont.
Lombardo, Thomas A., 2630 Gladys, Beaumont.
Lowry, Harvey M., 3155 Stagg Dr., Beaumont.
Lyons, Sam B., 801 Goodhue Bldg., Beaumont.
Makins, James, Medical Bldg., Port Arthur.
Mann, David A., (Hon.),
2135 Victoria St., Beaumont.
Mann, James C., 3280 Fannin St., Beaumont.
Manning, Wiley M., 2081 Park St., Beaumont.
Marroquin, Luis, 1632 7th Ave., Port Arthur.
Martin, John D., (Hon.),
1897 Rivercrest, Beaumont.
Matlock, Thos. B.,
1950 Ninth Ave., Port Arthur.
Matthews, Julius B.,
535 W. 13th St., Port Arthur.
McCall, John W., Jr.,
1785 Washington Blvd., Beaumont.
McNemer, Philip H.,
Perlstein Bldg. Annex, Beaumont.
McRee, Edgar C., Medical Bldg., Port Arthur.
McRee, Walter E., Jr.,
3040 Procter St., Port Arthur.
Meyer, Paul R., 3121 Procter, Port Arthur.
Miller, Kenneth T., 3155 Stagg Dr., Beaumont.
Mitchell, Theo C.,
2449 Calder Ave., Beaumont.
Montagnet, Joseph M., Jr.,
590 Center St., Beaumont.
Moore, Darwin D., 3155 Stagg Dr., Beaumont.
Moore, Robert E.,
1405 Nederland Ave., Nederland.
Newton, William A., 185 21st St., Beaumont.
Odell, Joyce W., (Inac.),
506 Goodhue Bldg., Beaumont.
Old, James R., 518 Goodhue Bldg., Beaumont.
Orgeron, Eugene B.,
541 Ninth Ave., Port Arthur.
Orrick, Robt. Jack, Jr.,
305 Doctors Bldg., Beaumont.
Orrill, R. Ray, Medical Bldg., Port Arthur.
Osborne, John A., Jr., 2525 Calder, Beaumont.
Pace, Bedford F., 555 Tenth St., Beaumont.
Painton, Clifford E.,
900 Ninth Ave., Port Arthur.
Pecora, Tony L.,
610 Goodhue Bldg., Beaumont.
Pentecost, Chas. L., 3155 Stagg Dr., Beaumont.
Petit, Paul T., 2627 Laurel Ave., Beaumont.
Petry, James L.,
3115 Procter, Port Arthur.
Phillippi, George M.,
2165 North St., Beaumont.
Pierson, Rogers, Box 2908, Beaumont.
Powell, James E., 3205 S. 4th St., Beaumont.
Pratt, George H.,
601 Goodhue Bldg., Beaumont.
Quick, David W., Jr.,
601 Goodhue Bldg., Beaumont.
Rafes, Earl H., 675 Fifth St., Beaumont.
Raines, James M.,
2002 Procter St., Port Arthur.
Reimers, Arthur F.,
Hotel Beaumont Bldg., Beaumont.
Richardson, Orville J.,
1405 Nederland Ave., Nederland.
Richman, Irving M., 3280 Fannin, Beaumont.
Robert, W. Pierre, 2630 Laurel Ave., Beaumont.
Rodick, John C., 2600 Procter St., Port Arthur.
Rollo, William C., Winnie.
Ross, Oliver H., 543 Ninth Ave., Port Arthur.
Rumelt, Allen 3504 Highland Ave., Beaumont.
Sappington, T. B., Jr.,
102 Dryden Place, Port Arthur.
Self, Milton Myrl
3155 Stagg Drive, Beaumont.
Serafino, Louis C.,
452 1st Fed. Sav. Bldg., Beaumont.
Sheldon, Robert B., 575 19th St., Beaumont.
Shorkey, Richard L., 370 Tenth St., Beaumont.
Shuffield, James W., Jr.,
3155 Stagg Drive, Beaumont.
Simpson, Rufus K.,
2372 Calder Ave., Beaumont.
Sladczyk, George, 3149 Procter, Port Arthur.
Smith, Denton W., 390 Tenth St., Beaumont.
Smith, Jack L., 1555 Driskill Dr., Beaumont.
Smith, John T., 203 Doctors Bldg., Beaumont.
Smith, William A.,
211 W. Caldwood Dr., Beaumont.
Solis, G. Robert, 2749 Procter St., Port Arthur.
Sprott, Curtis B., 2390 Cartwright, Beaumont.
Sprott, E. D., Jr., 2390 Cartwright, Beaumont.
Sprott, Maxie C., 2390 Cartwright, Beaumont.
Starr, Harry, 675 Fifth St., Beaumont.
Stephenson, G. Bruce,
370 Tenth St., Beaumont.
Stevens, Robert B.,
1297 Calder Ave., Beaumont.
Steward, Clyde A.,
2600 Procter St., Port Arthur.
Stewart, James K., P. O. Box 3311, Beaumont.
Stoeltje, Joe, 1170 Simmons St., Beaumont.
St. Romain, Chester J.,
1133 Boston Ave., Nederland
Suehs, Max E.,
228 San Jacinto Blvd., Beaumont.
Sutton, Fred W., Goodhue Bldg., Beaumont.
Sykes, Walter P., 3280 Fannin, Beaumont.
Tew, Alton H., 3155 Stagg Dr., Beaumont.
Thomas, James R., Gulf Oil Corp, Port Arthur.
Thompson, Douglas J.,
2600 Procter St., Port Arthur.

Thompson, James D.,
2600 Procter St., Port Arthur.
Todd, Charles H., 3155 Stagg Drive, Beaumont.
Toomim, Leonard A.,
3155 Stagg Drive, Beaumont.
Travis, John M., Jr.
3155 Stagg Drive, Beaumont.
Tritico, Joseph J., 949 7th St., Port Arthur.
Tyndall, Thomas M.,
2643 McFaddin, Beaumont.
Vaughan, Benjamin H.,
Adams Bldg., Port Arthur.
Walker, Charles R.,
502 Goodhue Bldg., Beaumont.
Walker, Taylor C., 2165 North St., Beaumont.
Wallace, Charles R., 860 Trinity, Beaumont.
Wallace, Loring Scott,
3155 Stagg Drive, Beaumont.
Wallace, Stuart M.,
1931 Ninth Ave., Port Arthur.
Wallace, William G.,
816 Goodhue Bldg., Beaumont.
Wallace, William V.,
5221 Procter St., Port Arthur.
Walters, Felix A.,
1133 Boston Ave., Nederland.
Walther, Charles D.,
P. O. Box 712, Port Arthur.
Washburn, Wesley W., Jr.,
3155 Stagg Drive, Beaumont.
Watts, Fenwick L.,
2220 Procter St., Port Arthur.
Weisbach, Philip T., 560 Center St., Beaumont.
Welch, Roland L.,
509 W. 10th St., Port Arthur.
Westbrook, Bonnie B., Jr.,
723 Washington, Beaumont.
White, Ben T., II,
1950 Ninth Ave., Port Arthur.
White, Clarence M.,
618 Amer. Natl. Bank Bldg., Beaumont.
White, J. Milton, Sr., (Inac.),
421 Fourth Ave., Port Arthur.
White, John M., Jr.,
3149 Procter St., Port Arthur.
Wier, Stuart T., 505 Goodhue Bldg., Beaumont.
Wilcox, Howard R.,
Hotel Dieu Hosp., Beaumont.
Williams, Chas. L., (Inac.),
1776 East Drive, Beaumont.
Williams, Ford G., 415 Center St., Beaumont.
Williams, Lewis M.,
3280 Fannin St., Beaumont.
Williams, Stewart M.,
3155 Stagg Drive, Beaumont.
Williford, Herman B.,
220 San Jacinto Blvd., Beaumont.
Willoughby, Russell C., Box 535, Groves.
Wilson, Francis W.,
P. O. Box 712, Port Arthur.
Woodward, John F., Jr.,
905 Goodhue Bldg., Beaumont.
Young, Isaac T., (Inac.),
P. O. Box 147, Port Arthur.

LIBERTY-CHAMBERS

Alexander, James H.,
P. O. Box 548, Cleveland.
Ashby, Joe M., Box 456, Anahuac.
Barnett, William L., P. O. Box 518, Cleveland.
Bellamy, Richard C., Box 1140, Liberty.
Castle, Charles W., 717 San Jacinto, Liberty.
Clements, Richard O., 715 Main, Liberty.
Davidson, Eli, Liberty.
Delaney, Albert L., Liberty.
Fahring, George H., Anahuac.
Fahring, Thomas L., Anahuac.
Gibson, Boyce E., Hull.
Griffin, Frank S., Liberty.
Jordan, Byron Lee, (Inac.), Daisetta.
Leggett, Walter, 106 N Bonham, Cleveland.
Neff, Louis E., 106 N. Bonham, Cleveland.
Parkman, Robert L., Jr., Box 1140, Liberty.
Richter, Ernest R., Dayton.
Schulz, Donald P., 608 Travis, Liberty.
Wilson, Reginald, Dayton.

NACOGDOCHES

Allen, James I., Nacogdoches.
Allen, Walter B., 1301 Raguet, Nacogdoches.
Beall, J. Frank, 2516 North St., Nacogdoches.
Buchele, Matthew J., Box R., San Augustine.
Coussons, Charles W., Box 482, Nacogdoches.
Ferguson, Sarah, 1301 Raguet, Nacogdoches.
Haley, Curtis R., Baggett Bldg., San Augustine.
Middlebrook, George F., (Inac.),
Rt. 5, Box 184, Nacogdoches.
Nelson, Albert L.,
422 E. Hospital St., Nacogdoches.
Neuville, Carroll F.,
P. O. Box 907, Nacogdoches.
Pennington, Thomas J.,
820 N. Mound, Nacogdoches.
Rogers, Eugene S., Nacogdoches.
Snider, L. W., Box G., Garrison.
Taylor, James G., Jr.,
1301 Raguet, Nacogdoches.
Tucker, F. Henry, Nacogdoches.
Tucker, Stephen B., Nacogdoches.
Williamson, Tom P.,
116 N Church St., Nacogdoches.

ORANGE

Allen, Rollie E., 1312 W. Park Ave., Orange.
Bennett, David 407 N. Ninth St., Orange.
Cloud, James P., 1212 W. Park Ave., Orange.
Covington, Chas., M.,
1005 W. Green Ave., Orange.
Eastman, George L.,
1005 W. Elm Ave., Orange.
Griffin, Oscar R., Box 750, Orange.
Harris, Mattice F., 1301 Second St., Orange.
Hume, Boyd Douglas, Jr.,
Box 258, Bridge City.
Ingram, Robert A., P. O. Box 479, Orange.
James, Wilson E., 1312 W. Park Ave., Orange.
Jirouch, Edwin A., 2202 McKee Drive, Orange.
Kent, Earl H., Box 2032, Orange.
Key, Harry H., Box 950, Orange.
Kokkinis, Hippocrates,
1107 W. Green, Orange.
McFadden, Irma M., P. O. Box 551, Orange.
Minkus, Robert F.,
2100 W. Yupon Rd., Orange.
Pearce, H. Wynne, 707 W. Main St., Orange.
Peters, Leo J., Jr., 601 N. 4th, Orange.
Pollock, Douglas H., 411 N. Ninth St., Orange.
Pretz, Earnest C.,
1604 N. Second St., Orange.
Schofield, Elmer C., (dead), Orange.
Seastrunk, Oliver C., 306 N. 10th, Orange.
Shaddock, Carroll B., 402 2nd St., Orange.
Siddon, William H., P. O. Box 2054, Orange.
Smith, Loyd C., Box 1205, Vidor.
Stewart, Jeannine E.,
2309 MacArthur Dr., Orange.
Stuntz, Billie W., 304 N. 7th, Orange.
Stuntz, Homer C., 304 N. 7th, Orange.
Thompson, Louis O., (Inac.),
121 W. 1st St., Biloxi, Miss.
Walsh, John K., 609 W. Park Ave., Orange.
Watson, Arthur R.,
2009 W. Coronado Pl., Orange.
Watson, Victor D., Box 1806, Vinton, La.
White, Malcolm E., 207 N. 7th St., Orange.
Williams, Howard C.,
1006 Green Ave., Orange.
Wyllie, James J.,
V. A. Hospital, Little Rock., Ark.

SHELBY-SAN AUGUSTINE-SABINE

Bridges, James P., Box 166, Center.
Hurst, Thomas L.,
503 Shelbyville St., Center.
Oates, Laried S., 116 Cora St., Center.
Warren, William H., P. O. Box 72, Center.
Warren, Wm. Spencer, Center.
Windham, John H., (Hon.), Shelbyville.
Windham, William C., P. O. Box 666, Center.

ELEVENTH DISTRICT

Dr. Robert H. Bell, Palestine, Councilor.

ANDERSON-HOUSTON-LEON

Bell, Robert H.,
109 E. Angelina St., Palestine.
Bentley, Bascom W., Jr.,
700 N. Sycamore St., Palestine.
Brown, Adelbert B., Jr., Box 942, Crockett.
Carter, J. Weldon, Palestine.
Cox, Robert G.,
804 S. Sycamore St., Palestine.
Darsey, Edward S., Box 820, Crockett.
Dean, John L., Jr., 407 East Lamar, Crockett.
Felder, Fred E., Palestine.
Goolsby, Carl B., Box 942, Crockett.
Griswold, Culver M., P. O. Box 672, Crockett.
Haverlah, Harry A., Palestine.
Humphries, John T., Palestine.
Hunter, Ripley H., Palestine.
Jordan, Curtis R., 103 Inwood Drive, Palestine.
Joyce, Claude D., Jr., Palestine.
Kay, Royal H., Palestine.
King, Marion A., Frankston.
Knowles, William R.,
205 E. Neches, Palestine.
Mathis, Lee Roy, Jr.,
107 Redland Lane, Palestine.
Mathis, Lucy L. E.,
107 Redland Lane, Palestine.
Murray, Carl O., Jr., Box 820, Crockett.
Powell, Elisha P., Centerville.
Prince, Homer E., Rt. 1, Box 109, Crockett.
Thompson, Jack R., 205 E. Neches, Palestine.
Van Wey, Archie E., Buffalo.
Wages, A. D., Palestine.
Walkup, Thomas E.,
804 S. Sycamore, Palestine.

CHEROKEE

Adams, Clyde, Box 31, Pineville, La.
Bailey, Ralph C.,
Matthewtown, Inagua, Bahamas.
Bilbro, Griff W., 1303 Hillcrest, Jacksonville.
Boyd, James T., 915 Hillcrest, Jacksonville.
Brigham, Floyd H., Rusk.
Chamness, Daniel E., Box 870, Jacksonville.
Cobble, Thomas H., 606 N. Main, Rusk.
Crawford, Joe D., Box 870, Jacksonville.
Davenport, Harbert, Jr.,
P. O. Box 1569, Jacksonville.
Fair, Harry E., Jr., Box 870, Jacksonville.
Gabbert, W. E., Rusk.
Hancock, Leslie D., P. O. Box 318, Rusk.
Hilliard, George M., Box 870, Jacksonville.
Jones, Ernest W., (dead), Rusk.
Kreimeyer, James H., Rusk State Hosp., Rusk.
Levin, Gus, Box 318, Rusk.
Mahon, Edward L., Jr., Box 870, Jacksonville.
Martin, Don E., Box 300, Wichita Falls.
Newburn, C. L., Box 1269, Jacksonville.
Noble, Roy C., Rusk State Hosp., Rusk.
Peek, John S., Box 318, Rusk.
Pryor, Victor W., 555 So. Jackson, Jacksonville.
Ralston, Leslie W., Box 870, Jacksonville.
Rives, Hugh F., Box 870, Jacksonville.
Roark, Wilson E., Box 870, Jacksonville.
Rossman, Robert E.,
4206 Tidewater Dr., Houston.
Rucker, J. Collier,
1117 Avalon Drive, Jacksonville.
Stacey, Royal K., Box 126, Castroville.
Stripling, C. H.,
555 S. Jackson St., Jacksonville.
Travis, John M., Box 870, Jacksonville.
Travis, L. L., Box 870, Jacksonville.
Travis, R. T., Box 870, Jacksonville.
Verheyden, Floyd H.,
813 John St., Jacksonville.

FREESTONE

Bonner, Leslie L., Box 157, Fairfield.
Buchmeyer, Norris D., P. O. Box 5, Wortham.
Cox, Jack R., Box 117, Teague.
Crossno, Joe D., Box 687, Fairfield.
Gage, Maurice, Box 278, Teague.
Halbert, Bill L., 315 Main St., Teague.
Keller, John H., Jr., Box 687, Fairfield.
Sneed, William N., (Hon.),
Box 103, Fairfield.

HENDERSON

Cockerell, Lonnie L., Athens.
Geddie, Nolen D., Jr., Athens.
Geddie, Nolen D., Athens.
Haynes, Clifford R., Kilman Hospital, Malakoff.
Henderson, Roy E., Athens.
Holt, Norris E., P. O. Box 188, Athens.
Kilman, Prather T., Malakoff.
Rosenbloom, Joseph, Trinidad.
Wilcox, Melvin R., 503 Bryson, Athens.

PANOLA

Baker, Charles D., (Hon.),
111½ N. St. Mary, Carthage.
Cooper, Grundy, 808 N. Daniel, Carthage.
Gerardy, Carl W., 317 Panola, Carthage.
Hargrove, Fred T.,
217 N. Pinewood Dr., Carthage.
Holland, Virgil M., 313 W. Panola, Carthage.
Johnson, Glen R., Box 818, Carthage.
Prince, Kenneth C.,
313 W. Panola St., Carthage.
Smith, William C.,
313 W. Panola St., Carthage.

RUSK

Braswell, Marlin T.,
119 S. Marshall, Henderson.
Deason, Loyd, 300 Ragley, Henderson.
Hamilton, James M., Box 908, Overton.
Heiligman, Haskell, Overton.
Hilbun, Lynn, 119 S. Marshall, Henderson.
Kennamer, Harold E., S. Helen St., Overton.
McShan, Chester L., 207B N. Main, Henderson.
Morris, Alfred E., 119 S. Marshall, Henderson.
Scaff, Martin C., 120 S. Marshall, Henderson.
Suehs, Herbert A.,
817 N. Marshall, Henderson.
Suehs, Paul E., 817 N. Marshall, Henderson.
Wolfe, Alfred S., 119 S. Marshall, Henderson.

SMITH

Alexander, J. Ernest, Jr., (Inac.),
214 E. Houston, Tyler.
Allen, George B., 818 Clinic Drive, Tyler.
Anderson, Carter, Jr., 712 S. Bois D'Arc, Tyler.
Avent, James K., 214 E. Houston, Tyler.
Bailes, Porter M., Jr.,
932 Hospital Drive, Tyler.
Baldwin, Russell E. G.,
1015 Hospital Drive, Tyler.
Bankhead, Alexander J.,
214 E. Houston, Tyler.
Baskin, T. Grady, 214 E. Houston, Tyler.
Betts, Floyd G., Jr., 1020 Clinic Drive, Tyler.
Birdwell, J. Weldon, 214 E. Houston, Tyler.
Blakemore, Henry C., 107½ E. Erwin, Tyler.
Bradford, Sidney W., 2015 S. College, Tyler.
Brelsford, H. Gates, 214 E. Houston, Tyler.
Bridges, Ben F., 214 E. Houston, Tyler.
Brown, George W., 305 W. Rusk St., Tyler.
Brown, Irving, 815 Peoples Bk. Bldg., Tyler.
Bryant, W. Howard, P. O. Box 600, Tyler.
Buie, Loyce C., 324 Amherst, Tyler.
Buie, Ralph R., 324 Amherst, Tyler.
Bundy, David T., (Hon.),
County Court House, Tyler.
Burch, George W., 908 Hospital Drive, Tyler.
Caldwell, Elbert H., 214 E. Houston, Tyler.
Calley, Clifford R., 1006 Clinic Drive, Tyler.
Cameron, Harold B., 214 E. Houston, Tyler.
Caplinger, Robert A., 1100 S. Beckham, Tyler.
Chenoweth, Carlin Velde,
1016 Clinic Drive, Tyler.
Clawater, Earl W., Jr.,
1016 Clinic Drive, Tyler.
Daniels, Herbert B., Jr.,
911 S. Beckham, Tyler.
DeCharles, Patrick M., 200 E. Houston, Tyler.
Duff, Robert L., 908 Hospital Drive, Tyler.
Edwards, Martin L.,
Edwards Clinic Hospital, Hawkins.
Etter, Roscoe, Arp.
Faust, John J., 903 S. Beckham, Tyler.
Ferrell, Oran L., Jr., (Int.),
Confederate Mem. Med. Center,
Shreveport, La.
Freiberg, Milton, 952 Hospital Drive, Tyler.
Gatti, Robert R., 1000 Clinic Drive, Tyler.
Goldfeder, Jesse, Box 2039, Tyler.
Goldsmith, J. Paul, 929 N. Glenwood, Tyler.
Goss, Vernon V., 908 Hospital Drive, Tyler.
Green, Frank D., 1012 E. Lake St., Tyler.
Halbrooks, Norman E., Box 3143, Tyler.
Hart, John Garrett, 217 W. Houston, Tyler.
Hughes, Delbert E., 900 Hospital Drive, Tyler.
Hughes, Waunell M.,
900 Hospital Drive, Tyler.
Jones, Joe C., 803 E. Houston, Tyler.
Knight, L. Roger, 538 Douglas Blvd., Tyler.
Lamberth, Ivey E., P. O. Box 3066, Tyler.
Lauck, Robert E., 1035 Clinic Drive, Tyler.
Lee, Madison J., Jr., 1100 S. Beckham, Tyler.
Lyles, Thomas W., 916 Hospital Dr., Tyler.
Marshall, Robert L., 1100 S. Beckham, Tyler.
McDonald Conrad C.,
900 Hospital Drive, Tyler.
McMillan, Bruce, (Inac), (dead), Overton.
Mitchell, John H., 901 S. Beckham, Tyler.
Moore, Masters H., 612 S. Bois D'Arc, Tyler.
Mullowney, James P.
926 Hospital Drive, Tyler.
Muntz, Hascall H., 938 Hospital Drive, Tyler.
Neill, J. Lawrence, 803 E. Houston, Tyler.
Neill, Lex T., 803 E. Houston, Tyler.
Payne, Donald E., 305 West Rusk, Tyler.
Pirtle, G. William, Jr.,
1000 Clinic Drive, Tyler.
Pope, Irvin, Jr., 118 S. Bois D'Arc, Tyler.
Rhine, Leland R., 911 S. Beckham, Tyler.
Rice, Elbert D., (Hon.), 434 S. Spring, Tyler.
Rice, Robert D., 1100 S. Beckham, Tyler.
Roosth, Wiley, 1025 Hospital Drive, Tyler.
Roper, Marjorie Ferrell, Bullard.
Rowe, Ralph W.,
Douglas Blvd. at E. Houston, Tyler.
Rowe, William E., 833 S. Beckham, Tyler.
Seaman, James B., Box 2003, Tyler.
Selman, Joseph, 1020 Clinic Drive, Tyler.
Shirley, T. Clayton, 911 S. Beckham, Tyler.
Smyth, William C., 1100 S. Beckham, Tyler.
Spence, Julius H., 3023 Jan Ave., Tyler.
Stephens, William C., 818 Clinic Drive, Tyler.
Stough, Dowling B., III,
1009 Hospital Drive, Tyler.
Taliaferro, Theodore J., 1106 Trezevant, Tyler.
Taylor, Phillip W., 833 S. Beckham, Tyler.
Thompson, Cone J., 810 Clinic Drive, Tyler.
Topperman, Samuel,
East Texas TB Hospital, Tyler.
Turner, John C., Jr., 1100 S. Beckham, Tyler.
Vaughn, Edgar H., 830 S. Beckham, Tyler.
Vaughn, James M., 830 S. Beckham, Tyler.
Warren, Frederick M., Jr.,
1012 E. Lake St., Tyler.
Whitten, Samuel J., Troup.
Wilcox, Leland G., 833 S. Beckham, Tyler.
Wiles, Jack, 1015 Hospital Drive, Tyler.
Williams, Frank E., 305½ N. Spring, Tyler.
Willingham, Chas. E. 911 S. Beckham, Tyler.
Wilson, Benjamin N., 1100 S. Beckham, Tyler.
Wimberley, Norris A., Jr.,
1001 S. Fleishel, Tyler.
Windham, Lynn B., Rt. 5, Box 110, Tyler.
Wood, James E., 929 N. Glenwood, Tyler.
Zuckerman, William, Box 2003, Tyler.

WOOD

Lane, Richard P., Quitman.
Mathis, James Ross, 408 S. Main, Winnsboro.
Merritt, Benjamin F.,
106 W. Bermuda, Quitman.
Moore, Roscoe O., 802 N. Pacific, Mineola.
Peterson, Thomas H., Mineola.
Reed, Theodore B., Mineola.
Robbins, Virgil E., Box 445, Quitman.
Williams, James W., Mineola.

TWELFTH DISTRICT

Dr. Tom M. Oliver, Waco, Councilor.

BELL

Alexander, John S., (Int.),
Scott & White Clinic, Temple.
Althaus, John W. A.,
McCloskey V. A. Hospital, Temple.
Anderson, Harold B.,
Scott & White Hospital, Temple.
Baden, Wayne F.,
Scott & White Clinic, Temple.
Bain, George P., Scott & White Clinic, Temple.
Bartels, Robert N., 304 S. 22nd St., Temple.
Bassel, Paul M.,
Scott & White Clinic, Temple.
Batch, Joseph W., (Mil),
U. S. Army Hospital, Fort Hood.
Best, Edward B., (Int.),
414 N.W. 36th Dr., Gainesville, Fla.
Bonnet, John D.
Scott & White Clinic, Temple.
Bradfield, Eldon O.,
Scott & White Clinic, Temple.
Brindley, George V.,
Scott & White Clinic, Temple.
Brindley, George V., Jr.,
Scott & White Clinic, Temple.
Brindley, Hanes H.,
Scott & White Clinic, Temple.
Broders, Albert C.,
Scott & White Clinic, Temple.
Broders, Albert C., Jr.,
Scott & White Clinic, Temple.
Broders, Charles W.,
Scott & White Clinic, Temple.
Brown, Jesse B.,
Kings Daughters Clinic, Temple.
Bruce, William J., 916 S. 45th, Temple.
Bryson, John B., Box 516, Belton.
Buchanan, Arren C., (Mil),
U. S. Army Hospital, Fort Hood.
Burow, F. Paul, Burow Clin. & Hosp., Killeen.
Bushey, Robert H., (Int.),
Scott & White Hospital, Temple.
Carabasi, Robert J.,
Scott & White Clinic, Temple.
Chernosky, William A.,
Kings Daughters Clinic, Temple.
Christian, John J.,
Scott & White Clinic, Temple.
Cochran, Leroy M., V. A. Center, Temple.
Cox, Charles H., Jr., 317 N. Second, Temple.
Cramm, Carl J., Jr.,
Scott & White Clinic, Temple.
Curtis, Raleigh R., 3617 W. 6th, Topeka, Kan.
Debord, Bert A., 2110 N. 15th, Temple.
Dysart, Donald N.,
Scott & White Clinic, Temple.
Eanes, David F. S., 518 N. 5th St., Temple.
Eiband, John M., (Int.),
Scott & White Clinic, Temple.
Ellis, Ira D., (Hon.), Troy.
Fillmore, Rollin S., Jr.,
Kings Daughters Clinic, Temple.
Ford, Joseph C., (Mil.),
USA Disp. 4052½, Fort Bliss.
Fowler, Joe A., 310 E. Ave B, Killeen.
Frey, Harry, (Inac),
107 Via Mentone, Newport Beach, Cal.
Gerdine, Park L.,
1402 E. Rancier Ave., Killeen.
Gillespie, Charles H.,
Scott & White Clinic, Temple.
Gober, Olin B., Scott & White Clinic, Temple.
Gordon, Grady C., V.A. Center, Temple.
Greenwood, Joseph H.,
Scott & White Hospital, Temple.
Grossman, Alex, V. A. Hospital, Temple.
Haines, Richard D.,
Scott & White Hospital, Temple.
Hamilton, Dixie G., Jr.,
710 W. Rancier, Killeen.
Hammond, Fred M., Jr.,
Scott & White Clinic, Temple.
Harlan, Rudolph K.,
Kings Daughters Hospital, Temple.
Hightower, Nicholas C.,
Scott & White Clinic, Temple.
Hill, Thomas L., V. A. Center, Temple.
Howell, Floyd W., (dead), Temple.
Ibarra, Jesse D., Jr.,
Scott & White Clinic, Temple.
Jarvis, Walter H., Jr.,
Scott & White Clinic, Temple.
Jenkins, Jesse G., (Hon.), Rt. 1, Temple.
Jones, Elmer A., V. A. Center, Temple.
Keil, Ernest W., 518 N. Fifth, Temple.
Kirkley, A. Rex, P. O. Box 240, Belton.
Laurens, Henry, Jr.,
Scott & White Clinic, Temple.
LaZarz, Donald T., (Int.),
Scott & White Clinic, Temple.
Lea, Walker A., Jr.,
Scott & White Clinic, Temple.
Lindsey, Sherman B.,
Scott & White Clinic, Temple.
Long, William B., 205 N. Pearl, Belton.
Lynch, Donald J.,
Scott & White Clinic, Temple.
McCelvey, John S., 304 S. 22nd St., Temple.
McKenney, John F., Jr.,
Scott & White Clinic, Temple.
McKibben, Byron G.,
Scott & White Clinic, Temple.
McRoberts, Marcus L.,
710 W. Rancier Ave., Killeen.
Milam, Robert W.,
Scott & White Clinic, Temple.
Millhon, William A., (Mil),
U. S. Army Hospital, Fort Hood.
Moore, James C., (Int.),
Scott & White Hospital, Temple.
Murray, Robert A.,
Scott & White Clinic, Temple.
Nelson, George C., (Int.),
Scott & White Hospital, Temple.
Padgett, John W., 310 Ave. B, Killeen.
Pettigrew, James A.,
Scott & White Hospital, Temple.
Pittman, John W., 325 N. Main, Belton.
Plasek, William W., 317 N. 2nd St., Temple.
Potter, Claudia, (Hon.),
707 S. 3rd St., Temple.
Powell, William N.,
Scott & White Clinic, Temple.
Robinson, E. Arthur, (Mil.),
U. S. Army Hospital, Fort Hood.
Rodarte, Jose G., Scott & White Clinic, Temple.
Ross, Warran A., (Int.),
Scott & White Hospital, Temple.
Schroeder, Henry F., (Mil.),
48th Med. Bn., Fort Hood.
Schubert, Herbert A., V. A. Hospital, Temple.
Sewell, Harvey W., Belton.
Sewell, Julian G., Belton.
Shelton, Walter B., Jr.,
317 N. 2nd St., Temple.
Sherman, Cyril F., V. A. Center, Temple.
Shibler, Samuel W.,
Kings Daughters Clinic, Temple.
Sommer, Arno W.,
Scott & White Hospital, Temple.
Speed, Terrell, Scott & White Hospital, Temple.
Spillar, Bliss R., Jr.,
Scott & White Clinic, Temple.
Spillar, Edna M. H., 2901 W. Ave. R, Temple.
Stinson, James C., 213 W. Ave. G, Temple.
Stokdyk, Glenn A., V. A. Center, Temple.
Sunbury, Thomas R.,
Scott & White Clinic, Temple.
Talley, J. Bartow, S. P. J. S. T. Bldg., Temple.
Talley, Lewis R.,
Kings Daughters Hospital, Temple.
Thompson, John Q.,
Scott & White Clinic, Temple.
Todd, James G., Jr., (Mil.),
U. S. Army Hospital, Fort Hood.
Veirs, Everett R.,
Scott & White Clinic, Temple.
Wahle, George Henry,
304 S. 22nd St., Temple.
Ward, Wendell P.,
912 1st Natl. Bk. Bldg., Temple.
Weeks, Bertram A., (Mil.),
U. S. Army Hospital, Fort Hood.
Weinblatt, Jack S., 518 N. 5th St., Temple.
Wells, Cora V., Rogers.
White, Raleigh R.,
Scott & White Clinic, Temple.

Whitten, Rondald J.,
1402 E. Rancier Ave., Killeen.
Williams, Bill Henry,
Scott & White Clinic, Temple.
Williams, Marjorie J., V. A. Hospital, Temple.
Wilson, James D.,
Kings Daughters Clinic, Temple.
Wolf, A. Ford, Scott & White Clinic, Temple.
Woodson, W. Burbank,
103 E. Central, Temple.

BOSQUE-HAMILTON

Blankenship, W. W., Mosheim.
Cleveland, Chas. C., Hamilton.
Gomez, Frank, Box 348, Evant.
Goodall, Van D., Clifton.
Holder, Wiseman T., Clifton.
Key, William F., Jr., 719 W. 11th, Clifton.
Kooken, Robert A., Hamilton.
Long, Austin M., Valley Mills.
Schadler, John A., 123 E. Henry, Hamilton.
Selman, Forrest B., 500 Rice St., Hamilton.
Witcher, Seth L., Clifton.

BRAZOS-ROBERTSON

Andres, Dwight W., 3501 Texas Ave., Bryan.
Baur, Paul S., 503 W. 28th St., Bryan.
Benbow, Robert H., 408 W. 28th St., Bryan.
Boyd, Elvin M., 1208 Live Oak, Hearne.
Cole, Charles M., Box 747, Bryan.
Cooper, James F., 308 W. 28th St., Bryan.
Cooper, Oliver C., 408 W. 28th St., Bryan.
Cox, Joseph M., 202 E. 27th St., Bryan.
Cruse, Ray, 1208 Live Oak, Hearne.
Fleming, James P., Jr., 710 Magnolia, Hearne.
Gallagher, Joseph C., Box 753, Hearne.
Geppert, Joseph W., 202 E. 27th St., Bryan.
Grant, Richard B., Jr., 308 W. 28th St., Bryan.
Harris, Nena A., 624 Mary Lake Drive, Bryan.
Harrison, R. Henry, 109 Porter Bldg., Bryan.
Harrison, Richard H., III,
109 Porter Bldg., Bryan.
Kemp, Clarence S., 905 Mitchell St., Bryan.
Lindsay, James I., 3501 Texas Ave., Bryan.
Marsh, John E., Jr., 701 S. College Ave., Bryan.
Marsh, John E., Sr., 3514 Cavitt St., Bryan.
Martin, Jay W., Jr., 2111 Vinewood, Bryan.
McGill, Albert G., 512 Varisco Bldg., Bryan.
McQuaide, Henry C., 3501 Texas, Bryan.
Melcher, Truman O., 202 E. 27th, Bryan.
Nelson, Kenneth L., Box 931, College Station.
Pope, Henry D., Jr., (Int.),
H. P. Long Char. Hospital, Pineville, La.
Reed, James E., Jr.,
603 S. Madison, Madisonville.
Richardson, S. C., (Hon.), Bryan.
Roman, William B., Jr.,
624 Mary Lake Drive, Bryan.
Russell, Robert M., 202 E. 27th St., Bryan.
Sanders, Jewell G., Bremond.
Searcy, R. M., Bryan.
Searcy, Thos. A., Hearne.
Smith, Roy L., 3501 Texas Ave., Bryan.
Taylor, William C., Jr., Calvert.
Tempesta, James E., 717 Broadmoor St., Bryan.
Walton, Thomas O., Box 434, College Station.
Walton, Thos. T., Bryan.
Wilkerson, Lonnie O., P. O. Box 1147, Bryan.
Willingham, J. Roy,
1208 Live Oak St., Hearne.

CORYELL

Brown, John T., 200 N. Lutterloh, Gatesville.
Jones, Kermit R., Gatesville.
Lowrey, E. Elworth,
116 N. Lutterloh, Gatesville.
Lowrey, O. Wendell, Gatesville.
Pack, Leonard C., 104 N. 7th St., Gatesville.

ERATH-HOOD-SOMERVELL

Altaras, Leon M., Box 2, Stephenville.
Bryan, T. F., (Hon.),
121 E. Black Jack, Dublin.
Cedars, Nathan,
441 N. Belknap St., Stephenville.
Cole, Charles T., 122 N. Grafton St., Dublin.
Hafer, Wm. Fred, Box 507, Hico.
Hearn, R. E., P. O. Box 513, Stephenville.
Hedges, Homer V., Hico.
Herrin, Bob J., Hico Clinic, Hico.
Jordan, Carl A., 109 N. Grafton St., Dublin.
Lancaster, Gus N., (Inac.), Granbury.
Pate, Joe J., 111 W. Black Jack, Dublin.
Reynolds, Avery M.,
DeLeon Mun. Hospital, DeLeon.
Richardson, Darwin L.,
405 S. Texas Ave., DeLeon.
Terrell, James C.,
411 N. Belknap, Stephenville.
Terrell, Vance, Box 371, Stephenville.
Terrill, Bruce S., 411 N. Belknap, Stephenville.
Wells, Buford A.,
411 N. Belknap, Stephenville.
Wier, David T., 120 Texas St., DeLeon.

FALLS

Barnett, John B., Torbett Clinic, Marlin.
Bennett, Alfred C., Torbett Clinic, Marlin.
Brown, James M., Torbett Clinic, Marlin.
Buie Neil D., Buie Clinic, Marlin.
Bussell, James S., Torbett Clinic, Marlin.
Cornwell, Charles H., Buie Clinic, Marlin.
Davison, Milton A.,
407 Chambers St., Marlin.
Giles, Roy G., (Hon.), 417 Houghton, Marlin.
Glass, Thomas G., Buie Clinic, Marlin.
Green, John E., Sr., 714 Coleman St., Marlin.
Hawkins, E. W., (Hon.),
120 Agarita Drive, Marlin.
Hughes, Sidney W., Torbett Clinic, Marlin.
Hutchings, Edgar P., Torbett Clinic, Marlin.
McDowell, Thomas A.,
229 Coleman St., Marlin.
McKinley, W. Frank, Jr.,
Torbett Clinic, Marlin.
Reese, Walter L., 407 Chambers St., Marlin.
Smith, Howard Lee, Torbett Clinic, Marlin.
Smith, Howard O., Torbett Clinic, Marlin.
Smith, Walter S., Torbett Clinic, Marlin.
Swepston, Happy J., Rosebud.
Swetland, Douglas R., Torbett Clinic, Marlin.
Useda, Domingo, 300 Coleman St., Marlin.

HILL

Barnes, Livingston, (Hon.), Hubbard.
Barton, James G., Whitney Clinic, Whitney.
Beskow, Richard N., 217 Craig St., Hillsboro.
Bradford, Andrew L., (Hon.), Coolidge.
Buie, James Morgan, 206 E. Elm, Hillsboro.
Campbell, Clark C., Itasca.
Cason, Dick K., 217 Craig, Hillsboro.
Garrett, Charles A., 300 Carr St., Hillsboro.
Grant, Silas W., 206 E. Elm, Hillsboro.
Guffy, Joseph L., Hillsboro.
Jenkins, Gaines H., (Hon.),
7629 Circle Dr., Lemon Grove, Calif.
Latham, John E., Jr., Whitney.
McDonald, Hendley A.,
206 N. Abbott St., Hillsboro.
Montgomery, John M.
206 E. Elm St., Hillsboro.
Morris, Thomas M., Mount Calm.
Sammons, Howard P., Hubbard.
Shirey, Robert W., Hillsboro.
Smith, Nellins C., 1107 Park Drive, Hillsboro.

JOHNSON

Allen, Charles G., 123 W. Main St., Itasca.
Ball, William P., Cleburne.
Barker, Gates R., 214 N. Caddo, Cleburne.
Bradford, Charles C., Box 21, Burleson.
Clark, Elmer L., 214 N. Caddo, Cleburne.
English, Robert D., Glen Rose.
Filardi, Hector H.,
310 W. Chambers, Cleburne.
Hamilton, Con D., Jr., Cleburne.
Hoehn, Bernice A., Box 218, Keene.
Jowell, Charlie C., Cleburne.
Kimbro, Robert W.,
310 W. Chambers, Cleburne.
Knox, Marshall T., Cleburne.
Little, John G., Cleburne.
Marks, Roger E., Glen Rose.
Peters, Joseph F., Jr., Box 431, Alvarado.
Pickens, J. Wendell, Cleburne.
Rice, John S., Jr., Kimbro Clinic, Cleburne.
Shiflett, Roland M., 624 N. Main, Cleburne.
Smyth, Olin T., Jr.,
111 S. Anglin St., Cleburne.
Thomas, Vernon L.,
Grandview Clinic, Grandview.
Whitehouse, Wm. R.,
310 W. Chambers, Cleburne.
Wiedenbauer, Margaret,
310 W. Chambers, Cleburne.
Wright, Glenn R.,
310 W. Chambers, Cleburne.
Yater, Tolbert F., Cleburne.

LIMESTONE

Barger, Marius I., 216 N. Sherman, Mexia.
Brewster, Charles H., 216 N. Sherman, Mexia.
Carrington, Wm. L., Mexia.
Christoffer, O. T., Mexia.
Cox, Stanley, Groesbeck.
Edgar, Cecil C., Mexia.
McKenzie, Casimir P., Mexia.
Standefer, Joe M., Mexia State School, Mexia.
Williford, Carl Ed, 216 N. Sherman, Mexia.
Wilson, Thelbert R., 311 N. Tyus, Groesbeck.

McLENNAN

Alexander, Boyd D.,
1105 Washington Ave, Waco.
Alexander, Robert B., 1105 Washington, Waco.
Allen, Homer B., Jr., 400 Main St., McGregor.
Anderson, Gilbert I., 1609 S. 12th St., Waco.
Anspach, Harold M., 1316 Austin Ave., Waco.
Atkins, Neal M., 605 Columbus St., Waco.
Avent, Woodrow M.,
1624 Colcord Ave., Waco.
Aynesworth, Horace T., (Hon.),
Box 157, Port Neches.
Aynesworth, M. Brian, 105 S. 18th St., Waco.
Barnes, Maurice C., 1310 Austin Ave., Waco.
Baskin, Roy H., Box 3278, Waco.
Bellegie, Nicholas J., Box 3278, Waco.
Berry, George W., 107 S. 18th St., Waco.
Braithwaite, Alfred T., 229 Clifton St., Waco.
Brewer, Jesse H., Jr., 3803 N. 22nd St., Waco.
Brooks, Cleveland H., (Hon.),
1104 Amicable Bldg., Waco.
Brothers, Joe F., 1205 N. 25th St., Waco.
Brown, Chadwick G., 211 N. Carpenter, Mart.
Brown, William W., Jr.,
2224 Washington, Waco.
Bryant, George C., (Int.),
1525 N. 15th St., Waco.
Burgess, John L., 1314 Austin Ave., Waco.
Butler, Jack, 3803 N. 22nd St., Waco.
Carlisle, Margil C., 1410 Austin Ave., Waco.
Carter, Marion I., 1905 N. 25th, Waco.
Catto, C. Gray, 1010 Professional, Waco.
Coffelt, Ralph L., 2320 Columbus, Waco.
Coleman, James A., Jr., 1901 N. 25th, Waco.
Colgin, James H., 2320 Columbus Ave., Waco.
Colgin, Merchant W.,
2320 Columbus Ave., Waco.
Collins, C. T., 2320 Columbus Ave., Waco.
Collins, Lawrence D., 2226 Washington, Waco.
Connally, H. Frank, 1716 Colcord Ave., Waco.
Corbett, Joseph H., 2226 Washington, Waco.
Crosthwait, R. Wilson, 107 S. 18th St., Waco.
Crosthwait, Wm. L., (Hon.),
107 S. 18th St., Waco.
Cunningham, Perry J.,
1806 Austin Ave., Waco.
Dudgeon, Howard R., Jr.,
2226 Washington, Waco.
Dulaney, Charles H., Box 3265, Waco.
Dunlap, John C., 2320 Columbus Ave., Waco.
Fadal, Richard G., 2425 Ethel Ave., Waco.
Fenno, Richard M., (Mil.),
4317 N. 19th St., Waco.
Fine, Eldon B., Professional Bldg., Waco.
Flowers, Jack W., 2524 Washington, Waco.
Ford, Walter L., V. A. Hospital, Waco.
Forsythe, John R., 115 S. 18th St., Waco.
Foy, James D., 1609 S. 12th St., Waco.
Friedman, Carl, 604 Med. Arts Bldg., Waco.
Friedman, Leo, 2505 Washington, Waco.
Gamble, Charles E., 2524 Washington, Waco.
Gandy, William M., 1710 Colcord Ave., Waco.
Garrett, James M., 1408 Austin Ave. Waco.
Gassler, Robert K., 1710 Colcord, Waco.
Gidney, William H., West.
Goodman, Aubrey L., 2216 Austin Ave., Waco.
Granger, W. Hanson, 1316 Austin Ave., Waco.
Gunter, Marion B., Mart.
Hanks, Robert J., 702 Prof. Bldg., Waco.
Harrell, Joe, 1806 Austin Ave., Waco.
Harris, Edwin B., 1316 Austin Ave., Waco.
Hejtmancik, James H.,
2313 Washington, Waco.
Hipps, Herbert E., 1612 Columbus Ave., Waco.
Hoehn, F. William,
2320 Columbus Ave., Waco.
Howard, Stanley P., 312 S. 8th St., Waco.
Husbands, Tom L. 1914 Colcord Ave., Waco.
Jaworski, H., 425 Austin Ave., Waco.
Johnson, Ernest A., 1710 Colcord, Waco.
Kee, John L., 805 Amicable Bldg., Waco.
King, Walter B., Jr.,
2320 Columbus Ave., Waco.
Kingsbery, Lloyd B.,
75 White Oak St., Franklin, N. C.
Klatt, Wesley W., Hillcrest Hospital, Waco.
Kleiman, Richard B., 1612 Colcord Ave., Waco.
Kochman, Walter P.,
1717 Columbus Ave., Waco.
Lattimore, John E. Amicable Bldg., Waco.
Locke, Corbet C., Jr., Box 3157, Waco.
MacRae, Malcolm M., 1905 N. 25th, Waco.
Magid, Moreton A., 1525 Colcord Ave., Waco.
Manske, Arnold O., 1917 Austin Ave., Waco.
Marstaller, Wm. E., Box 3265, Waco.
McMahan, George T., V. A. Center, Waco.
Mewshaw, R. E. L., (Hon.),
1717 S. 9th St., Waco.
Milam, E. A., (Hon.),
1412 Austin Ave., Waco.

Miller, Claire F., Box 1338, Waco.
Mitchell, Holland C., V. A. Hospital, Waco.
Montgomery, Hazel I., (Int.),
3724 N. Versailles, Dallas.
Morrison, S. Bergen, 2317 Washington, Waco.
Murphey, Paul C., 2320 Columbus Ave., Waco.
Nichols, Ace E., 2508 Austin, Waco.
Oliver, Tom M., 1722 Colcord Ave., Waco.
Power, Paul H., (Hon.),
1011 Prof. Bldg., Waco.
Reese, Clarence H., Professional Bldg., Waco.
Richey, Harvey M., 1722 Colcord Ave., Waco.
Robertson, Lester E.,
416 W. 2nd St., McGregor.
Roche, Buell F., 711 Prof. Bldg., Waco.
Roddy, William N., 115 S. 18th St., Waco.
Ross, Philip H., 3323 Brook Circle, Waco.
Rottner, Mark H., 2223 Austin Ave., Waco.
Sadler, Leslie R., 2320 Columbus Ave., Waco.
Scanio, Thomas J.,
3321 Hillcrest Drive, Waco.
Schwartze, Erich W., 1609 S. 12th St., Waco.
Scruggs, James H., Jr.,
2223 Austin Ave., Waco.
Shellenberger, C. G., 2524 Washington, Waco.
Shipp, John R., Professional Bldg., Waco.
Shipp, W. R. F., (Hon.), Lorena.
Simpson, Neill O., 111 S. 18th St., Waco.
Slade, Harry W., 2224 Washington, Waco.
Sloan, Jack Q., 1901 N. 25th, Waco.
Smith, C. Collom, 1804 Austin Ave., Waco.
Souther, William L., (Hon.),
811 Prof. Bldg., Waco.
Spark, Milton, 121 Dallas, Waco.
Spencer, Shelby C., (Hon.),
Professional Bldg., Waco.
Spencer, W. Bruce, 2226 Washington, Waco.
Stanislav, F. J., (Hon.), 1820 Colonial, Waco.
Stewart, Howard R.,
3001 Bellmead Drive, Waco.
Tabb, K. Stewart, 1616 Colcord Ave., Waco.
Tabb, T. Edgar, Co. Health Dept., Waco.
Talley, John E., 2317 Washington, Waco.
Thompson, John E., McGregor.
Traylor, Clayton J.,
2320 Columbus Ave., Waco.
Trippet, Horace H.,
2320 Columbus Ave., Waco.
van den Sigtenhorst, Andries,
2320 Columbus Ave., Waco.
Wagnon, William E., Jr.,
2220 Austin Ave., Waco.
Warren, Daniel D., 1722 Colcord Ave., Waco.
Weekley, F. Clay, Box 3147, Waco.
Wilson, Loyal K., 1726 Colcord Ave., Waco.
Wittstruck, Kenneth P.,
Providence Hospital, Waco.
Wood, James K., 2505 Morrow Ave., Waco.
Wood, R. Spencer, (Hon.),
Professional Bldg., Waco.
Woodward, Joseph W.
2220 Austin Ave., Waco.
Woolsey, Fleta G., 210 N. 7th St., Waco.
Woolsey, Henry U., 210 N. 7th St., Waco.
Woolsey, William J., 210 N. 7th St., Waco.
Wybourn, Robert C.,
1316 Austin Ave., Waco.

MILAM

Barkley, Thomas S., Box 1487, Rockdale.
Crump, Thos. E., Cameron.
Green, Travis C.,
730 W. Cameron St., Rockdale.
Halbert, Horace B., Jr., Box 513, Rosebud.
Hamilton, Lawrence E., Rogers.
Newton, William R., Cameron.
Richards, John T., 602 N. Main St., Rockdale.
Richardson, Sidney H.,
P. O. Drawer 270, Cameron.
Selden, Lawrence E., 602 N. Main, Rockdale.
Shapiro, David, Cameron.
Smith, Elmer O., Jr., Cameron.
Swift, Clifford G., Cameron.
Young, Philip M., 602 N. Main, Rockdale.

NAVARRO

Barnebee, James H., Jr.,
205 South 15th, Corsicana.
Bone, Robert D., Box 841, Corsicana
Brown, Charles H., Jr.,
401 W. Collin, Corsicana.
Burnett, Samuel H., (Hon.),
Box 168, Corsicana.
Campbell, C. David, 205 S. 15th St., Corsicana.
Campbell, Robert L., 205 S. 15th St., Corsicana.
Carter, William W., (Hon.),
328 S. 31st St., Corsicana.
David, J. Wilson, 401 W. Collin, Corsicana.
Egyed, Miklos H., Box 527, Corsicana.
Gary, Charles L., Jr., Box 633, Corsicana.
Gibson, Louis E., Box 841, Corsicana.
Griffin, Ben H., Frost.
Griffin, John W., Box 841, Corsicana.
Grizzaffi, Anthony L.,
205 S. 15th St., Corsicana.
Hamill, Dan B., Box 1356, Corsicana.
Hansford, David P.,
Med. Arts Clinic, Corsicana.
Karn, Willard R., Jr., Box 841, Corsicana.
Kelton, Leslie E., Jr., Box 583, Corsicana.
Logsdon, William K., Box 598, Corsicana.
Mayfield, William B.,
205 S. 15th St., Corsicana.
McGary, Lester E., Jr.,
401 West Collin, Corsicana.
McGary, Lester E., Sr.,
Navarro Co. Mem. Hosp., Corsicana.
Megarity, Andrew L., Jr.,
413 N. 38th St., Corsicana.
Mertz, Robert D., Box 841, Corsicana.
Miller, Will M., Box 1135, Corsicana.
Mitchell, Paul H.,
218 W. Second Ave., Corsicana.
Neese, George E., Frost.
Newton, Earl H., (Hon.), Box 394, Corsicana.
Pannill, Fitzhugh C., Jr.,
Hahnemann Med. Col., Philadelphia, Pa.
Rosen, Bernard, 1205 Columbia Cir., Corsicana.
Sanders, Gurley H., Kerens.
Sanders, Ivan T., Kerens.
Shell, William T., Jr., Box 527, Corsicana.
Sneed, William R., 401 W. Collin, Corsicana.

THIRTEENTH DISTRICT

Dr. Travis Smith, Abilene, Councilor

BAYLOR-KNOX-HASKELL

Balch, Edwin H., Seymour.
Bunkley, E. P., (Hon.), Stamford.
Edwards, T. S., Knox City.
Eiland, David C., Munday.
Frizzell, Thos. P., Knox City.
Markward, Chas. G.,
Knox City Clinic, Knox City.
Massa, Joseph A.,
407 N. Washington, Seymour.
Middleton, Robert A., Haskell.
Newsom, Robert L., Munday.
Randal, Chas. M., Jr., Seymour.
Rogers, Madison W., (Hon.), Rule.
Scott, Frank C., Haskell.
Taylor, William M., (Hon.), Goree.
Williams, Temple W., Haskell.

CLAY-MONTAGUE-WISE

Bryant, David W., Box 428, Bridgeport.
Crumpler, Hulen P., Bowie.
Crumpler, Prentice, Jr., Bowie.
Darwin, James T., Decatur.
Harris, Ewing P., Bowie Clin. Hosp., Bowie.
Huddleston, William E.,
Bridgeport Hosp., Bridgeport.
Hurn, Robert E., Henrietta.
Inabnett, W. T., Decatur.
Major, A. D., Box 239, Nocona.
Major, John W., Box 239, Nocona.
Merritt, Ben C., Jr.,
Rogers Hospital, Decatur.
Pesek, Edward F., (Mil.),
437 Surg. Hosp., San Francisco, Calif.
Rogers, Thomas G., Decatur.
Squyres, Berry N., Tarrant at Lindsey, Bowie.
Strickland, George E.,
316 West Omega, Henrietta.
Valcik, John H., Decatur.
Wharram, Kenneth J., Box 207, Nocona.

EASTLAND-CALLAHAN-STEPHENS-SHACKELFORD-THROCKMORTON

Addy, Ervin E., Jr., 1106 W. 6th, Cisco.
Alexander, Billy Bob, Petroleum Bldg., Eastland.
Ball, D., Cisco.
Ballew, Charles H., 900 W. 8th St., Cisco.
Bradley, Ben H., Rising Star Hosp., Rising Star.
Brogdon, William M.,
Blackwell Hosp., Gorman.
Brown, Audie A., Gorman.
Bulgerin, Harold J.,
507 Connelle St., Eastland.
Caton, James H., (Hon.),
700 W. Moss St., Eastland.
Goodall, Edwin, 109 E. Dyer, Breckenridge.
Graham, Emmett L., Cisco.
Howle, Thomas M., Post Office Bldg., Albany.
Lawrence, Jim T., Breckenridge.
Mims, Douglas L., 211 N. Austin, Ranger.
Mitchell, Otis C., 3875 Kenneth St., Beaumont.
Payne, Frank C., 111 S. Miller, Breckenridge.
Stubblefield, M. L., Box 1046, Baird.
Townsend, Edwin R., Eastland.
Treadwell, Mose A., Jr.,
404 W. Commerce, Eastland.
Watkins, Wirter P.,
% The Ranger Cl., Ranger.
Whittington, James C., Eastland.

PALO PINTO-PARKER-YOUNG-JACK-ARCHER

Abney, Thomas B., 1016 S. Main, Weatherford.
Allen, Platt L., Weatherford.
Allensworth, John C.,
208 NW 2nd St., Mineral Wells.
Allensworth, William B.,
208 NW 2nd St., Mineral Wells.
Conner, Paul K., Drawer 2, Jacksboro.
Divine, Blaine, Graham.
Eidson, Jack L.,
614 Palo Pinto St., Weatherford.
Gafford, Gordon M.,
415 NW 2nd St., Mineral Wells.
Gowan, R. E. L., Graham.
Griffin, B. B., Graham.
Griffin, H. E., Graham
Jordan, Jack, 237 W. Archer, Jacksboro.
Kenner, Joanne,
208 NW Second St., Mineral Wells.
Lasater, Waldo B., Box 77, Mineral Wells.
Mask, William G., 237 W. Archer, Jacksboro.
McCall, James D.,
Baker Hotel Bldg., Mineral Wells.
McCloud, Ben L., Mineral Wells.
McClure, C. C., 108 S. Seventh, Jacksboro.
McDaniel, Robert G., Box 209, Graham.
McKinney, Hugh C., (Hon.), Olney.
Merrick, John B., Weatherford.
Nelson, Joe T., Weatherford.
Oates, K. D., Graham.
O'Quin, William A.,
Baker Hotel Bldg., Mineral Wells.
Padgett, W. O., Graham.
Parker, Philip J., 805 Cherry St., Graham.
Patterson, Andrew M., Box 70, Mineral Wells.
Pedigo, Paul C., Strawn.
Roan, John L., 106 South Main, Weatherford.
Roberson, John F., (Hon.), Gordon.
Rosser, V. O., Jr., 614 Third St., Graham.
Russell, Earl M., Weatherford.
Smith, John E., Box 248, Weatherford.
Smith, Robert H., Palo Pinto.
Spears, Jean Wiley, Graham.
Whalen, Carl H., Weatherford.
Wheelis, Brewer D., Drawer 2, Jacksboro.
Williams, Charles R., Mineral Wells.
Yeager, Edward F.,
Baker Hotel Bldg., Mineral Wells.

TARRANT

Adams, Kenneth P.,
1300 Fielder Rd., Arlington.
Alliston, Wiley S., Jr.,
701 5th Ave., Fort Worth.
Anderson, Herman W., Jr.,
612 Eighth Ave., Fort Worth.
Andujar, John J.,
2951 Benbrook Blvd., Fort Worth.
Anthony, Ernest E., Jr.,
409 Med. Arts. Bldg., Fort Worth.
Anthony, Walter P., Jr.,
705 Fifth Ave., Fort Worth.
Archer, Maurice C.,
800 Fifth Ave., Fort Worth.
Armstrong, Wm. F.,
1400 8th Ave., Fort Worth.
Aurin, Fred B.,
1216 Pennsylvania, Fort Worth.
Auringer, Arthur J., 410 W. Abram, Arlington.
Austin, Carl M., 1325 S. Main, Fort Worth.
Avery, Jack W., 515 S. Summit, Fort Worth.
Axtell, Earl C., (Hon.),
5741 Electric Ave., LaJolla, Calif.
Bailey, Noel R.,
1506 W. Terrell St., Fort Worth.
Baker, Robert G.,
715 Med. Arts Bldg., Fort Worth.
Ball, Bert C., 604 Doctors Bldg., Fort Worth.
Ballinger, T. Irwin,
3309 N.E. 28th St., Fort Worth.
Banker, Harry W., Box 1719, Fort Worth.
Barcus, James R.,
708 S. Henderson, Fort Worth.
Barcus, Wm. Shelton,
708 S. Henderson, Fort Worth.
Barker, Robert C., 1501 Summit, Fort Worth.
Barnes, Chas. K.,
Neil P. Anderson Bldg., Fort Worth.
Barrett, Isaac P.,
3210 W. Lancaster, Fort Worth.
Barrier, Chas. W.,
Medical Arts Bldg., Fort Worth.
Bean, George W., 715 Fifth Ave., Fort Worth.
Beard, Bruce H., 921 Eighth Ave., Fort Worth.
Beasley, C. Harold
1201 W. Presidio, Fort Worth.

Beasley, Grace A., Box 9223, Fort Worth.
Beaton, Hugh,
1316 Med. Arts Bldg., Fort Worth.
Begley, Grant F.,
1415 Pennsylvania, Fort Worth.
Bell, J. Ardis,
7211 Grapevine Hwy., Fort Worth.
Bendel, Henry W., Jr.,
607 E. Abram, Arlington.
Bennett, Jerrell, 650 S. Henderson, Fort Worth.
Bertram, Harold F.,
650 Fifth Ave., Fort Worth.
Bibby, Douglas E.,
1217 W. Cannon, Fort Worth.
Bickel, Robert D.,
Professional Bldg., Fort Worth.
Bida, John F., 715 W. Abram, Arlington.
Binion, Warren W.,
1201 W. Presidio, Fort Worth.
Birdsong, Karl K.,
4012 Harlanwood Dr., Fort Worth.
Birdwell, Henry V., Jr.,
1501 Summit, Fort Worth.
Bishop, Alton A.,
2501 W. Berry St., Fort Worth.
Black, Thos. W., 808 8th Ave, Fort Worth.
Blaha, Frank J.,
2404 W. Seminary Dr., Fort Worth.
Bobo, Zack, Jr., 311 S. Center, Arlington.
Bolch, Alfred P.,
413 4th St., N.W., Independence, Iowa.
Boles, Truett C.,
600 W. Park Row, Arlington.
Boluch, John J., 121 W. Pipeline Rd., Hurst.
Bond, Tom B.,
815 Med. Arts Bldg., Fort Worth.
Bonelli, Victor E., (Inac.),
611 W. 4th St., Fort Worth.
Bontley, Paul T., 909 Briarwood, Arlington.
Booth, Earnest, St. Josephs Hosp, Fort Worth.
Boren, Darrell W.,
3341-A Winthrop, Fort Worth.
Borg, Fritz H., 800 Fifth Ave., Fort Worth.
Borum, Valington F.,
312 Crestwood Dr., Fort Worth.
Boston, Don Wayne,
4012 S. Freeway, Fort Worth.
Bowden, Andy J., Jr.,
650 S. Henderson, Fort Worth.
Boyd, Craig H., 800 Fifth Ave., Fort Worth.
Bradshaw, Wilber V., Jr.,
1800 University Dr., Fort Worth.
Braselton, Chas. W., Jr.,
1050 W. Dashwood, Fort Worth.
Brasher, Ray V.,
1000 W. Petersmith, Fort Worth.
Brentlinger, Robert W.,
1005 Sherwood, Arlington.
Brock, Bill L., 307 New York, Arlington.
Brock, Ernest H., (Inac.),
707 Hawkins, Fort Worth.
Bronstad, Morris T., Jr.,
3101 Winthrop, Fort Worth.
Brooks, Donald A.,
2200 Evans Ave., Fort Worth.
Brooks, James W., 5182 Ollie, Fort Worth.
Brooks, Marion J.,
2200 Evans Ave., Fort Worth.
Brous, Marion W.,
1012 S. Henderson, Fort Worth.
Brown, Arthur, 1015 Lamar, Fort Worth.
Brown, Clarence E.,
209 Waters St., Mansfield.
Brown, Robert W., 811 Fifth Ave, Fort Worth.
Brown, W. Porter,
1106 Med. Arts Bldg., Fort Worth.
Brownfield, Jack D.,
806 S. Lake St., Fort Worth.
Bruner, Cavan B., 510 S. Ballinger, Fort Worth.
Burgess, Richard M.,
3226 E. Rosedale, Fort Worth.
Burnett, Jesse M., 415 East Sixth, Fort Worth.
Bursey, Leroy 3101 Winthrop, Fort Worth.
Bussey, Joe L., 1201 W. Presidio, Fort Worth.
Bussey, Thomas B.,
1101 Med. Arts Bldg., Fort Worth.
Butler, Alan W., Jr.,
1001 5th Ave., Fort Worth.
Bynum, Frank L., 650 Fifth Ave., Fort Worth.
Cameron, Robert P.,
800 Fifth Ave., Fort Worth.
Campbell, J. Franklin,
1410 Pruitt, Fort Worth.
Campbell, Ralph E., (Mil.),
Ireland Army Hosp, Fort Knox, Ky.
Caroe, A. Edward, 611 E. Abram, Arlington.
Carpenter, Nathan C.,
Fort Worth Natl. Bank Bldg., Fort Worth.
Carr, Robert E., 800 Fifth Ave., Fort Worth.
Carter, Peter J.,
6721 Telephone Rd., Fort Worth.
Carter, Willis H., 800 Fifth Ave., Fort Worth.
Cayce, John H.,
1015 Pennsylvania, Fort Worth.
Chalmers, William D.,
306 W. Broadway, Fort Worth.
Chambers, James O.,
1519 Pennsylvania, Fort Worth.
Chapman, Martha J.,
5808 E. Belknap, Fort Worth.
Chapman, Robert E.,
5808 E. Belknap, Fort Worth.
Childs, Tilden L., Jr.,
3102 Greene, Fort Worth.
Chorn, Etheredge H.,
554 S. Summit, Fort Worth.
Church, John M.,
1221 W. Lancaster, Fort Worth.
Claunch, DeWitt, 900 Eighth Ave., Fort Worth.
Clayton, Chas. F.,
1100 W. Cannon, Fort Worth.
Clayton, Irvin, 1100 W. Cannon, Fort Worth.
Clifford, William S.,
1017 Pennsylvania, Fort Worth.
Cochran, J. Robert, 717 5th Ave., Fort Worth.
Cohen, Frank, 712 S. Henderson, Fort Worth.
Cohn, Robert L., 312 W. Hurst Blvd., Hurst.
Coleman, Thos. J.,
1221 W. Lancaster, Fort Worth.
Collier, Julius W.,
4012 S. Freeway, Fort Worth.
Colvin, Joseph W.,
5201 Camp Bowie Blvd., Fort Worth.
Compere, Dolphus E.,
1415 Pennsylvania, Fort Worth.
Conner, Cooper M.,
1055 West Terrell, Fort Worth.
Cook, Percy L., 209 Waters, Mansfield.
Cook, Willis G., (Hon.),
3625 Park Hill Dr., Fort Worth.
Council, Francis E.,
1404 Pennsylvania, Fort Worth.
Council, Frank C., Jr.,
705 Randol Mill Rd., Arlington.
Crabb, McKinley, H.,
1710 Med. Arts Bldg., Fort Worth.
Crawford, William M.,
612 Sixth Ave., Fort Worth.
Cristol, David A., 800 Fifth Ave., Fort Worth.
Cross, Thomas J.,
1414 Pennsylvania, Fort Worth.
Cunningham, Ernest S.,
921 8th Ave, Fort Worth.
Daly, Jack E., (Hon.),
2200 Huntington, Fort Worth.
Darnell, Ruth Little,
3125 Handley Dr., Fort Worth.
Daugherty, Frank J.,
515 South Summit, Fort Worth.
Davenport, Emory, Box 1719, Fort Worth.
Davis, Charles P., 918 Eighth Ave., Fort Worth.
Day, Giles W., 910 Eighth Ave., Fort Worth.
Deaton, Hobart O., 616 Sixth Ave., Fort Worth.
DeBusk, Jack S.,
1001 Pennsylvania, Fort Worth.
Dees, Doyce B., Jr., (Mil.),
3812 Kimberly Lane, Fort Worth.
Dingwerth, Frank S.,
1300 Fielder Rd, Arlington.
Ditto, Hugh H., 510 S. Ballinger, Fort Worth.
Doss, A. Keller, 800 Fifth Ave., Fort Worth.
Doss, Doyle J., 3427 E. Lancaster, Fort Worth.
Douthit, Thomas E., Jr.,
2327 N. Riverside, Fort Worth.
Duffy, Jack G.,
3750 S. University, Fort Worth.
Dunn, Nelson L., (Inac.),
Medical Arts Bldg., Fort Worth.
Duringer, William C., (Hon.),
3729 Lenox Drive, Fort Worth.
Edwards, Walter T.,
4104 E. Lancaster, Fort Worth.
Ellis, Joe B., (Inc.),
1300 W. Cannon, Fort Worth.
Emery, Oscar J., Box 1926, Fort Worth.
Eschenbrenner, John W.,
Vet. Adm. Center, Shreveport, La.
Estes, Ben P., 900 Eighth Ave., Fort Worth.
Etier, Edgar L., Jr.,
650 Fifth Ave., Fort Worth.
Ezell, Edgar S., Box 1719, Fort Worth.
Fain, Harold B., P. O. Box 100, Fort Worth.
Farley, James A., 1000 Fifth Ave., Fort Worth.
Farris, Chester A., Jr.,
1832 E. Abram, Arlington.
Fershtand, John B., 1501 Summit, Fort Worth.
Fitzwilliam, C. Dennis,
800 Fifth Ave., Fort Worth.
Flood, William E.,
Medical Arts Bldg., Fort Worth.
Funk, Theron H., 501 S. Summit, Fort Worth.
Furman, John M., Jr.,
404 Med. Arts Bldg., Fort Worth.
Gambill, Alice F.,
5410 Camp Bowie, Fort Worth.
Gardiner, Henry G., Jr.,
Box 1719, Fort Worth.
Garnett, John W., Jr.,
1016 W. Cannon, Fort Worth.
Gentling, Allen A., 5109 Bryce, Fort Worth.
Gibbs, Donald S., 650 Fifth Ave., Fort Worth.
Gilliland, Lloyd N., Jr.,
424 S. Summit, Fort Worth.
Goatcher, Joann H., 5309 Waits, Fort Worth.
Godley, Louie O., (Hon.),
2129 Park Place, Fort Worth.
Goggans, Albert M., 811 5th Ave., Fort Worth.
Goldberg, Abraham I.,
Burk Burnett Bldg., Fort Worth.
Goldberg, Morton N.,
Burkburnett Bldg., Fort Worth.
Goldman, Winfrey W., Jr.,
703 W. Randol Mill Rd., Arlington.
Gooch, Frank B.,
1009 Pennsylvnaia, Fort Worth.
Gracia, Valentin,
1408 Med. Arts Bldg., Fort Worth.
Grammer, James H.,
1400 8th Ave., Fort Worth.
Grammer, Richard B.,
1400 8th Ave., Fort Worth.
Gray, Billy N., 1517 E. Berry, Fort Worth.
Greines, Abe, 1549 N. Main St., Fort Worth.
Greve, Anna M.,
1210 Virginia Pl., Fort Worth.
Grice, Thomas W.,
1312 Med. Arts Bldg., Fort Worth.
Griffin, Anderson A.,
1137 E. Bessie, Fort Worth.
Griffin, Otho P., 800 Fifth Ave, Fort Worth.
Grogan, Oscar R., 3200 Avondale, Fort Worth.
Grogan, Roy L., 921 Fifth Ave., Fort Worth.
Grunow, Otto H., 650 5th Ave., Fort Worth.
Guinn, Edward W., 5306 Ramey, Fort Worth.
Guinn, James E.,
614-A Missouri Ave., Fort Worth.
Guy, Robert S., 1001 Pennsylvania, Fort Worth.
Gwozdz, Feliks, P. O. Box 1719, Fort Worth.
Haffke, Oscar W.,
1017 Pennsylvania, Fort Worth.
Hageman, Phyllis M.,
3461 Blue Bonnet, Fort Worth.
Haggard, Fred A., (Hon.),
Westbrook Hotel, Fort Worth.
Hall, Ewin P., Jr.,
402 Med. Arts Bldg., Fort Worth.
Hallmark, James A.,
951 W. Magnolia, Fort Worth.
Halpin, Frank W., 3500 W. 7th, Fort Worth.
Hardey, C. Eugene,
600 W. Park Row, Arlington.
Harding, Robert J.,
3513 Mansfield Rd., Fort Worth.
Hardwick, B. Randol,
3341-A Winthrop, Fort Worth.
Hardwick, Jack F.,
3341-A Winthrop, Fort Worth.
Harle, Harold L.,
1300 W. Cannon, Fort Worth.
Harris, J. Robert,
1115 Pennsylvania, Fort Worth.
Harrison, Edwin S.,
2014 N. Riverside, Fort Worth.
Hartnett, Dalton C.,
800 Fifth Ave., Fort Worth.
Hatch, Garth S., 508 W. Park Row, Arlington.
Hawker, Laverne J.,
1556 W. Magnolia, Fort Worth.
Hawkins, Chas. P., 1524 Pruitt St., Fort Worth.
Hayes, Charles F., (Hon.),
1609 Harrington, Fort Worth.
Head, William M., 1325 S. Main, Fort Worth.
Heberle, James P., P. O. Box 597, Burleson.
Heinrichs, Andrew N.,
800 Fifth Ave., Fort Worth.
Helbing, Hugh V.,
1549 N. Main St., Fort Worth.
Hendrix, Wm. C., Jr.,
800 Fifth Ave., Fort Worth.
Hewatt, John W.,
3301 E. Rosedale, Fort Worth.
Hicks, Chas. E., 3101 Winthrop, Fort Worth.
Hiett, Carey 815 5th Ave., Fort Worth.
Higgins, Wm. P., Jr.,
501 Med. Arts Bldg., Fort Worth.
Hill, Robert D., 712 S. Henderson, Fort Worth.
Hoehn, David, 800 Fifth Ave., Fort Worth.
Holmes, James E.,
1606 Med. Arts Bldg., Fort Worth.
Holmgren, Robert B., 1501 Pruitt, Fort Worth.
Holsapple, Cortell K.,
1221 W. Lancaster, Fort Worth.
Hooker, James F., III,
1111 Pennsylvania, Fort Worth.
Hopkins, Donald H.,
2100 Hwy. 183, N.W., Fort Worth.
Hopkins, Glenn A.,
301 W. Central, Fort Worth.

Horn, William S., Jr.,
650 Fifth Ave., Fort Worth.
Howard, Rex J., 930 Fifth Ave., Fort Worth.
Howard, Rex Z., Box 865, Fort Worth.
Huffman, Andrew M.,
1201 Hemphill St., Fort Worth.
Hulsey, Sim, 701 5th Ave., Fort Worth.
Hutcheson, George O.,
1216 Pennsylvania, Fort Worth.
Innis, John J., 914 W. Cannon St., Fort Worth.
Irvine, James, Jr., 2212 Carleton, Fort Worth.
Isaacks, Hub E.,
1415 Pennsylvania, Fort Worth.
Isbell, Marney C.,
2129 Robinwood, Fort Worth.
Jackson, Atras E., (Hon.),
3204 S. University, Fort Worth.
Jackson, Holland T.,
1602 Med. Arts Bldg., Fort Worth.
Jacobson, Bruce K.,
7211 Grapevine Hwy., Fort Worth.
Jagoda, Samuel, 1501 Summit, Fort Worth.
Jenkins, Wesley N.,
3102 Greene Ave., Fort Worth.
Jermyn, John W.,
6717 Grapevine Hwy., Fort Worth.
Jernigan, C. Richard,
708 E. Abram, Arlington.
Jernigan, John M.,
1101 W. Berry, Fort Worth.
Jernigan, Lane M.,
1101 W. Berry, Fort Worth.
Jewell, George W., Jr.,
5201 River Oaks Blvd., Fort Worth.
Johnson, Clive R., 800 Fifth Ave., Fort Worth.
Johnson, F. Melvin,
7112 Camp Bowie, Fort Worth.
Johnston, Dewey W.,
901 W. Leuda St., Fort Worth.
Jones, Shellie J., Jr., (Mil.),
8304 Saddle Trail, Fort Worth.
Jordan, Carl F.,
1800 University Dr., Fort Worth.
Jorns, Kenneth L.,
424 S. Summit, Fort Worth.
Kauffmann, Adolph F.,
707 Fifth Ave., Fort Worth.
Keith, Joseph M., 501 S. Summit, Fort Worth.
Kelly, Gordon B.,
1001 Pennsylvania, Fort Worth.
Kerr, Walter C. H.,
1209 Med. Arts Bldg., Fort Worth.
Key, Robert E., 915 Fifth Ave., Fort Worth.
Kibbie, Horace K.,
2417 Medford Ct. E., Fort Worth.
Kibbie, Kent V., (Hon.),
Med. Arts Bldg., Fort Worth.
King, Gerald A.,
3951 E. Lancaster, Fort Worth.
Kingsbury, Herman B.,
316 Med. Arts Bldg., Fort Worth.
Kleuser, Lawrence P.,
930 Fifth Ave., Fort Worth.
Knox, Warren G.,
2905 Vaughn Blvd., Fort Worth.
Kramer, John T., Jr.,
5050 E. Belknap, Fort Worth.
Kyger, E. Ross, Jr., 800 Fifth Ave., Fort Worth.
Lace, W. Ted, 1001 Pennsylvania, Fort Worth.
Lacy, Geo. W.,
515 Med. Arts Bldg., Fort Worth.
Ladd, Arnett D.,
306 W. Broadway, Fort Worth.
Lancaster, Edgar L.,
1011 West NW Hwy., Grapevine.
Lancaster, Minnie L.,
1011 West NW Hwy., Grapevine.
Lange, Arthur A.,
508 Med. Arts Bldg., Fort Worth.
Lankford, Frances L. P., (Int.),
2306 Welborn, Dallas.
Lansford, Doyle K.,
1958 E. Park Row, Arlington.
Lauderdale, Thos. L.,
1307 Pennsylvania, Fort Worth.
Leaffer, Harry, 800 Fifth Ave., Fort Worth.
Lee, David F.,
3513 Mansfield Hwy., Fort Worth.
Lees, Chas. R.,
805 Med. Arts Bldg., Fort Worth.
Lemon, Robert G., 811 5th Ave., Fort Worth.
Lenox, Walter R., 800 Fifth Ave., Fort Worth.
Leon, William R.,
1001 Pennsylvania, Fort Worth.
Leverett, Jack K., Convair, Fort Worth.
Levy, Louis J., 918 Eighth Ave, Fort Worth.
Lindsay, John F., Jr.,
800 Fifth Ave., Fort Worth.
Lipscomb, Cuvier P.,
918 Eighth Ave., Fort Worth.
Littlepage, Henry B.,
1503 Med. Arts Bldg., Fort Worth.
Looney, Robert H., Jr.,
1505 Med. Arts Bldg., Fort Worth.
Lorimer, Wishard S., (Hon.),
3851 Winslow Dr., Fort Worth.
Lorimer, Wishard S., Jr.,
901 W. Leuda, Fort Worth.
Luke, Edward A.,
3112 Handley Dr., Fort Worth.
Lyle, Judge M., 510 S. Ballinger, Fort Worth.
Machado, Gaston, 510 W. Park Row, Arlington.
Maddox, Stephen G., Jr.,
5216 W. Freeway, Fort Worth.
Mallard, Robert S.,
1216 Pennsylvania, Fort Worth.
Malone, James D.,
424 S. Summit, Fort Worth.
Marietta, John S.,
1115-B Pennsylvania, Fort Worth.
Marrow, William S.,
1519 Pennsylvania, Fort Worth.
Marts, Walford D.,
306 W. Broadway, Fort Worth.
Marshall, Al K.,
1800 University Dr., Fort Worth.
Matheson, Dan N.,
Medical Arts Bldg., Fort Worth.
Maxwell, Earl, P. O. Box 1959, Fort Worth.
Mayo, Talmadge D., Jr.,
921 5th Ave., Fort Worth.
McBride, James O.,
1405 Med. Arts Bldg., Fort Worth.
McCarroll, Molloy C.,
2501 Forest Park, Fort Worth.
McClelland, Gene A., 2812 Yates, Fort Worth.
McDaniel, Thos. W., Jr.,
1410 Med. Arts Bldg., Fort Worth.
McDonald, Earl D.,
1320 S. Collins, Arlington.
McDonald, Henry C., Jr.,
918 8th Avenue, Fort Worth.
McDonald, Robert P.,
1010 W. Rosedale, Fort Worth.
McKee, France A., Jr.,
305 Prof. Bldg., Fort Worth.
McKee, Frank, 1016 W. Cannon, Fort Worth.
McKee, Frank S.,
1016 W. Cannon, Fort Worth.
McKee, Richard,
1016 W. Cannon, Fort Worth.
McKenzie, Walten H.,
800 S. Lake St., Fort Worth.
McKinney, William W.,
800 Fifth Avenue, Fort Worth.
McKnight, W. Hodges,
Amon Carter Field, Fort Worth.
McVeigh, Joseph F.,
306 W. Broadway, Fort Worth.
Melch, Herbert R., Jr.,
3805 Southwest Blvd, Fort Worth.
Mitchell, D. Gatlin,
1604 Med. Arts Bldg., Fort Worth.
Mitchell, Jerrell L.,
3800 Camp Bowie Blvd., Fort Worth.
Monaghan, Johnnie E., Jr.,
1009 Pennsylvania, Fort Worth.
Moore, Louis G., 1320 S. Collins, Arlington.
Moorman, Warren W.,
901 W. Leuda, Fort Worth.
Moreton, Robert D.,
1217 W. Cannon, Fort Worth.
Morgan, William H.,
1109 Pennsylvania, Fort Worth.
Morphis, Oscar L.,
815 Med. Arts Bldg., Fort Worth.
Morris, Abner J., 3432 W. 7th St., Fort Worth.
Murchison, St. Julien R.,
803 Med. Arts Bldg., Fort Worth.
Murphy, James D.,
1556 W. Magnolia, Fort Worth.
Mycoskie, Bernard J.,
3rd & Southwest, Arlington.
Myers, Theodore B.,
800 Fifth Avenue, Fort Worth.
Myers, William T.,
Westcliff Center, Fort Worth.
Neal, Durwood E.,
1556 W. Magnolia, Fort Worth.
Needham, Robert H., (Hon.),
Continental Life Bldg., Fort Worth.
Neighbors, DeWitt, 701 5th Ave., Fort Worth.
Neil, William H.,
815 Med. Arts Bldg., Fort Worth.
Nesbit, Preston M., 309 E. Main, Arlington.
Nyman, Randall D.,
1015 Pennsylvania, Fort Worth.
O'Bannon, Roscoe P.,
1216 Pennsylvania, Fort Worth.
Olcott, Eugene D., 425 S. Summit, Fort Worth.
O'Reilly, John J., (Hon.),
19 Peak Road, Austin.
Ott, William O.,
1017 W. Terrell Ave., Fort Worth.
Owen, May, Box 1719, Fort Worth.
Patterson, John B., 800 5th Ave., Fort Worth.
Pena, Heriberto,
NE 28th & Schwartz, Fort Worth.
Perkins, Lynn C.,
3851 S.W. Loop, Fort Worth.
Petra, Walter B.,
1556 W. Magnolia, Fort Worth.
Phillips, Oliver M.,
1221 W. Lancaster, Fort Worth.
Phillips, William G., 3021 Race, Fort Worth.
Pierce, John R.,
705 Randol Mill Rd., Arlington.
Pillow, David J.,
7211 Grapevine Hwy., Fort Worth.
Potts, John (Hon.),
301 Danciger Bldg., Fort Worth.
Price, Earl P., Jr.,
2501 Forest Park, Fort Worth.
Price, Richard V., 1325 S. Main, Fort Worth.
Price, Sidney A.,
1216 Pennsylvania, Fort Worth.
Prine, John M., 1300 Fielder Rd., Arlington.
Prudhomme, Eck G., Jr.,
5153 River Oaks Blvd., Fort Worth.
Pumphrey, Andrew B.,
1701 Med. Arts Bldg., Fort Worth.
Ramey, Paul M.,
1615 Med. Arts Bldg., Fort Worth.
Rank, Dale C., 607 E. Abram, Arlington.
Rape, Joe G., 715 W. Abram, Arlington.
Rapfogel, Irving,
1414 W. Humbolt, Fort Worth.
Rathgeber, Van D.,
1305 Med. Arts Bldg., Fort Worth.
Readinger, Ivan H.,
1000 Fifth Ave., Fort Worth.
Reeves, Ernest E.,
1507 Med. Arts Bldg., Fort Worth.
Reeves, Leopold H., (Emer.),
1016 W. Arlington, Fort Worth.
Rehfeldt, Frederick C.,
1001 Pennsylvania, Fort Worth.
Renshaw, Horace S.,
914 W. Cannon, Fort Worth.
Richards, John H.,
4222 Birchman St., Fort Worth.
Riddell, J. Murray, Jr.,
800 Fifth Ave., Fort Worth.
Riddle, Charles H.,
3116 Dreeben, Fort Worth.
Riley, Jack C.,
1425 Pennsylvania, Fort Worth.
Rimmer, Raymond J.,
1009 Pennsylvania, Fort Worth.
Risley, Thomas W.,
5153 River Oaks Blvd., Fort Worth.
Roan, Leo N., 4727 Camp Bowie, Fort Worth.
Robertson, Alexander D.,
Box 1719, Fort Worth.
Robinson, Charles, 912 Fifth Ave., Fort Worth.
Robinson, Irvin, 912 Fifth Ave., Fort Worth.
Rogers, Ernest D., 515 S. Summit, Fort Worth.
Rogers, R. Cromwell, (Hon.),
Hilton Hotel, Fort Worth.
Rohrer, Vern 800 Fifth Ave., Fort Worth.
Ross, Nealie E., 701 5th Ave., Fort Worth.
Rubin, Norman L.,
4220 Harlanwood, Fort Worth.
Rumph, Mal, 705 Med. Arts Bldg., Fort Worth.
Rush, Charles A., Jr., Box 13112, Fort Worth.
Rutledge, Art H., Box 1719, Fort Worth.
Rutledge, Robb H.,
800 Fifth Avenue, Fort Worth.
Satterwhite, Raymond D.,
5182 Ollie St., Fort Worth.
Savage, Hugh W., 815 Fifth Ave., Fort Worth.
Savage, James Munroe, Box 538, Azle.
Schaffer, Richard C., Box 1719, Fort Worth.
Scharff, Earle U., Jr.,
904 Burk Burnett Bldg., Fort Worth.
Schenck, Chas. P., (Hon.),
1012 Med Arts Bldg., Fort Worth.
Schoolfield, Emmett C., (Inac.),
4637 Pershing, Fort Worth.
Schoonover, Frank S., Jr.,
608 S. Adams, Fort Worth.
Schwarz, Edwin G.,
712 S. Henderson, Fort Worth.
Scroggie, Val D.,
1001 Pennsylvania, Fort Worth.
Sealy, Wm. Burgess,
800 Fifth Ave., Fort Worth.
Sehested, Herman C.,
815 Med. Arts Bldg., Fort Worth.
Sewell, John H.,
1221 W. Lancaster, Fort Worth.
Sewell, Robert L.,
1221 W. Lancaster, Fort Worth.
Sheddan, Frank G. (Inac.),
1313 Sinclair Bldg., Fort Worth.
Sheinberg, Philip, 400 W. Hurst Blvd., Hurst.
Shelley, Harold J.,
1000 Fifth Ave., Fort Worth.
Shields, Thos. L.,
1216 Pennsylvania, Fort Worth.

Shilling, Harold C.,
2100 Hwy. 183, N.W., Fort Worth.
Shoemaker, Thos. J. W.,
Stockyd. Natl. Bldg., Fort Worth.
Short, James W., 811 Fifth Ave., Fort Worth.
Siddons, Geo. Y.,
1307 Pennsylvania, Fort Worth.
Silva, Samuel, 1301 N. Main, Fort Worth.
Skokan, William, Med. Arts Bldg., Fort Worth.
Small, David E., 911 Sixth Ave., Fort Worth.
Smith, G. Con., 650 Fifth Ave., Fort Worth.
Smith, J. Ronald,
1607 Med. Arts Bldg., Fort Worth.
Smith, Jack C., 800 Fifth Ave., Fort Worth.
Smith, Stanley C.,
1216 Pennsylvania, Fort Worth.
Snyder, Frank L.,
304 Virginia Pl., Fort Worth.
Snyder, Roy E.,
203 Med. Arts Bldg., Fort Worth.
Spackman, Edgar W., (Inac.),
General Delivery, San Juan.
Stadler, Earl W., 800 Fifth Ave., Fort Worth.
Starr, Pete J., 607 E. Abram, Arlington.
Steeg, Galen E., 715 W. Abram, Arlington.
Steger, Joseph H.,
923 Pennsylvania, Fort Worth.
Steinberger, Eugene,
901 W. Cannon, Fort Worth.
Stewart, George A., Jr.,
1015 Pennsylvania, Fort Worth.
Stouffer, James G.,
1506 W. Terrell Ave., Fort Worth.
Stout, Sidney E., 650 Fifth Ave., Fort Worth.
Stow, Robert C., Jr.,
306 W. Broadway, Fort Worth.
Sumner, Wendell W., 600 Bailey, Fort Worth.
Swift, William B., 1501 Summit, Fort Worth.
Swords, H. Logan,
301 W. Central Ave., Fort Worth.
Tadlock, Marvin E.,
Burk Burnett Bldg., Fort Worth.
Tatum, William C.,
1304 Med. Arts Bldg., Fort Worth.
Taylor, Elbert D., 5534 E. Belknap, Fort Worth.
Taylor, Elizabeth A.,
Box 1719, Fort Worth.
Taylor, Thomas U., II,
1009 Pennsylvania, Fort Worth.
Teague, William H.,
4420 NE 28th St., Fort Worth.
Terrell, Blanche O.,
1425 Pennsylvania, Fort Worth.
Terrell, Caleb O., Jr.,
1000 Fifth Ave., Fort Worth.
Terrell, Chas. J.,
602 W. 10th St., Fort Worth.
Terrell, Truman C., Box 1719, Fort Worth.
Thomas, Henry W., Jr.,
1612 NW 28th St., Fort Worth.
Thomas, Hiram C.,
1055 W. Terrell, Fort Worth.
Thomason, Thos. H.,
1226 Pennsylvania, Fort Worth.
Thompson, Burl V.,
1400 8th Ave., Fort Worth.
Tipps, D. Conway,
900 Eighth Ave., Fort Worth.
Toppin, Bruce E.,
3427 E. Lancaster, Fort Worth.
Tottenham, John W., (Hon.),
4016 W. 6th St., Fort Worth.
Tottenham, John W., Jr.,
3706 Tulsa Way, Fort Worth.
Touzel, Cecil S. E.,
1111 Pennsylvania, Fort Worth.
Tucker, John T.,
404 West 3rd St., Fort Worth.
Tucker, John T., Jr.,
404 West 3rd St., Fort Worth.
Tunstill, James W.,
1501 Summit, Fort Worth.
Turner, Jack L.,
515 S. Summit, Fort Worth.
Van Zandt, Isaac L., 915 5th Ave., Fort Worth.
Viard, Walter S., Jr.,
800 Fifth Avenue, Fort Worth.
Walker, James N.,
5216 W. Freeway, Fort Worth.
Walker, Webb,
703 Med. Arts Bldg., Fort Worth.
Wallace, E. Frank, 715 W. Abram, Arlington.
Wallace, John L., Jr.,
P. O. Box 11435, Fort Worth.
Walsh, Edmund N.,
1410 Pruitt, Fort Worth.
Ware, Drue O. D.,
1302 W. Magnolia, Fort Worth.
Watson, Asa C., Jr.,
815 5th Ave., Fort Worth.
West, Walter B.,
951 W. Magnolia, Fort Worth.
Westfall, Cad B., 1501 Summit, Fort Worth.
White, James B.,
1300 W. Cannon, Fort Worth.
White, Richard J.,
W. T. Waggoner Bldg., Fort Worth.
Wier, Edward M.,
1401 W. Rio Grande, Fort Worth.
Wiggins, J. Kenneth,
800 Fifth Avenue, Fort Worth.
Wiggins, John A.,
1105 Pennsylvania, Fort Worth.
Willess, Hersel, F., 800 Fifth Ave., Fort Worth.
Williams, Claude, 622 E. Abram, Arlington.
Williams, Marion F.,
1017 Pennsylvania, Fort Worth.
Willis, Selwyn A.,
Third & Southwest, Arlington.
Willis, Virginia G.,
2208 Timberline Dr., Fort Worth.
Wilson, Edwin B., Jr.,
1501 Summit, Fort Worth.
Wilson, Stephen W.,
914 Med. Arts Bldg., Fort Worth.
Winterringer, James R.,
800 Fifth Ave., Fort Worth.
Wise, Joe R.,
1519 Pennsylvania, Fort Worth.
Witt, Roger W.,
312 Crestwood Dr., Fort Worth.
Wollenman, Oscar J., Jr.,
1724 Martel, Fort Worth.
Womack, Harry H.,
1424 W. Peter Smith, Fort Worth.
Womble, Joe D.,
1300 Fielder Rd., Arlington.
Wood, William W., Jr.,
811 Med. Arts Bldg., Fort Worth.
Woodward, Cicero S.,
Knights Templar Hosp., Arlington.
Woodward, Valin R., (Inac.),
P. O. Box 1172, Austin.
Worrall, Cyrus L., 800 5th Ave., Fort Worth.
Wyss, Herbert E., Keller.
Zimmerman, Ben E.,
4720 E. Lancaster, Fort Worth.

TAYLOR-JONES

Adams, Clinton E., 1436 N. 4th St., Abilene.
Adamson, William B.,
1434 N. 4th St., Abilene.
Ailts, Bernard H., 4755 Hartford, Abilene.
Anderson, F. Katherine,
1026 N. 21st St., Abilene.
Andrus, Allen G., Pittard Bldg., Anson.
Arrant, Arthur G., 868 Hickory St., Abilene.
Barker, Frank R.,
1237 N. Mockingbird, Abilene.
Barron, Stanton J.,
1241 N. Mockingbird, Abilene.
Bessire, Milton C., 1325 Hickory St., Abilene.
Bowyer, Mack F., 1101 N. 19th St., Abilene.
Boyd, Virginia H., Box 333, Abilene.
Bray, Willis J., 1325 Hickory St., Abilene.
Bridge, Harry R.,
1229 N. Mockingbird, Abilene.
Bunkley, Thomas A., Stamford San., Stamford.
Burditt, Jesse N., Clinic Bldg., Abilene.
Burditt, Tom C., 1442 N. 3rd St., Abilene.
Burns, Coleman C., 1026 N. 21st St., Abilene.
Buzbee, H. Ray, 1101 N. 19th St., Abilene.
Cadenhead, James F., Haskell.
Cash, W. Auda V., (Inac.),
901 Albany St., Abilene.
Chapel, James P.,
1442 N. 3rd St., Abilene.
Clark, J. Frank, 1229 S. 7th St., Abilene.
Cochran, William E., P. O. Box 451, Abilene.
Cockerell, Earl R., Clinic Bldg., Abilene.
Colbert, Robert E., Rule Clinic, Rule.
Crow, Jack A., 1825 Hickory, Abilene.
Dawson, George A.,
1235 N. Mockingbird, Abilene.
Day, Wm. Kenneth, 4755 Hartford, Abilene.
Domm, Bill M., 1325 Hickory, Abilene.
Donnell, Ralph E., Jr.,
1133 N. 19th St., Abilene.
Duff, J. C., 1054 12th St., Anson.
Estes, Bobby Jack, 641 Hickory St., Abilene.
Estes, Jack M., 641 Hickory, Abilene.
Estes, Sol B., 1502 N. 2nd St., Abilene.
Evans, Robert W., Clyde.
Fox, W. Irby, 1227 N. Mockingbird, Abilene.
Gardner, Chester B., Merkel.
Gibson, John P., 202 Grape St., Abilene.
Guerra, Jose J., Box 451, Abilene.
Hamilton, Hinton H.,
4755 Hartford St., Abilene.
Harper, Orville E., 1101 N. 19th St., Abilene.
Hawkins, Elmer J., Hamlin.
Haynes, Jack S., 725 Leggette Dr., Abilene.
Hollis, Scott W., 1725 Hickory St., Abilene.
Hooks, Jim M., 1133 N. 19th St., Abilene.
Hudson, F. E., (Hon.), Stamford.
Hudson, Isaac F., Stamford.
Jensen, Meredith N.,
1101 N. 19th St., Abilene.
Johns, Richard B., 802 Mulberry St., Abilene.
Johnson, Dale F., 4755 Hartford, Abilene.
Johnson, Laurence F., Clinic Bldg., Abilene.
Magee, Jefferson D., Jr.,
1101 N. 19th St., Abilene.
McDonald, Donald H., Clinic Bldg., Abilene.
McFadden, C. Alfred,
1325 Hickory St., Abilene.
Merrick, J. Estes, 851 Orange St., Abilene.
Metz, Louis F., (dead), Stamford.
Middleton, Edwin E., 1442 N. 3rd St., Abilene.
Morgan, Clyde N., 2073 Cedar, Abilene.
Murtha, Carroll E., 3333 S. 11th St., Abilene.
Nystrom, Robert E., 1442 N. 3rd St., Abilene.
Palasota, Pete C., 254 Leggett Dr., Abilene.
Pate, Virgil A., Jr., 1101 N. 19th St., Abilene.
Pattillo, Guy L., 2073 Cedar St., Abilene.
Perrin, E. Douglas, Hamlin.
Petty, Preston D., 1325 Hickory, Abilene.
Pittard, Knox J., 1054 12th St., Anson.
Pope, Andrew J., (Inac.),
1854 S. 7th St., Abilene.
Porterfield, D. G.,
1231 N. Mockingbird, Abilene.
Prichard, Clarence L., (Inac.),
2042 S. 8th, Abilene.
Pruet, Royce W., 1325 Hickory, Abilene.
Pryor, George E., Jr., Stamford.
Pugh, David F.,
426 N. Terrace Dr., Wichita, Kansas.
Ramsey, Wayne V., 1725 Hickory St., Abilene.
Ramsey, Wayne V., Jr.,
1101 N. 19th St., Abilene.
Rode, R. Lee Henry, 1101 N. 19th St., Abilene.
Roe, Maurice A., 2241 S. 19th St., Abilene.
Sadler, William T., Merkel.
Seale, Hubert J., 1325 Hickory, Abilene.
Seale, W. Hubert, 1325 Hickory, Abilene.
Sellers, Erle D., Clinic Bldg., Abilene.
Selmon, Tony B., Stamford.
Shoultz, V. H., 1101 N. 19th St., Abilene.
Sibley, William R., Jr.,
1101 N. 19th St., Abilene.
Smith, Marshall L., Hamlin.
Smith, Travis, 1442 N. 3rd St., Abilene.
Snow, Joseph C., 851 Orange, Abilene.
Steward, Mary Booth, 773 Cypress, Abilene.
Strole, Donald G., 1026 N. 21st, Abilene.
Taylor, Floyd D., 4755 Hartford, Abilene.
Thigpen, Joe E., 1417 N. 1st St., Haskell.
Thurman, George D.,
1101 N. 19th St., Abilene.
Trotter, Billy Bob,
Hendrick Mem. Hosp., Abilene.
Tull, Raymond H., 1142 N. 13th St., Abilene.
Turnbull, Marshall D.,
1026 N. 21st St., Abilene.
Varner, Roy W., 1026 N. 21st St., Abilene.
Warren, Donald W.,
Sadler Clinic Hospital, Merkel.
Webster, L. J., 1101 N. 19th St., Abilene.
Williams, Charles F., 116½ Chestnut, Abilene.
Williams, Jarrett E., 1101 N. 19th St., Abilene.
Williams, William H.,
1325 Hickory St., Abilene.
Williamson, Lee, 4755 Hartford, Abilene.

WICHITA

Acker, Julian H.,
1300 8th St., Wichita Falls.
Adams, Jerome M.,
121 Hill Top, Burkburnett.
Adams, Walter B., Jr.,
1100 8th St., Wichita Falls.
Adams, Walter B., Sr.,
1100 8th St., Wichita Falls.
Alexander, Howard G.,
Wichita State Hosp., Wichita Falls.
Allen, David H.,
Medical Arts Bldg., Wichita Falls.
Arrington, John H.,
1411 9th St., Wichita Falls.
Bates, Charles R.,
1501 5th St., Wichita Falls.
Bates, Harriet H., (Int.),
Wichita Gen. Hosp., Wichita Falls.
Bebb, Edwin C., 5th & Broad, Wichita Falls.
Bebb, Kenneth C., 1518 10th St., Wichita Falls.
Berg, Owen C., 1300 8th St., Wichita Falls.
Bondurant, Dewry C.,
4811 Lake Park Dr., Wichita Falls.
Bradford, Clarence T., (Hon.),
1404 Hayden, Amarillo.
Brown, Charles H.,
1418 Eighth St., Wichita Falls.
Browne, Frank S.,
Medical Arts Bldg., Wichita Falls.
Buchanan, Martha B., 700 Brook, Wichita Falls.
Burleson, James W.,
2502 Tenth St., Wichita Falls.
Burross, D. Clifford,
4007 Faith Road, Wichita Falls.

Carpenter, Philip A., Burkburnett.
Carson, John D., 1518 Tenth St., Wichita Falls.
Caskey, Marion W.,
311 Hamilton Bldg., Wichita Falls.
Clark, Gordon G., Iowa Park.
Coleman, Blair P.,
1300 8th St., Wichita Falls.
Collins, R. Paul, 500 Broad St., Wichita Falls.
Cook, Hulen J., Jr., 1500 8th St., Wichita Falls.
Cox, E. Aubrey, 5th & Broad Sts., Wichita Falls.
Crump, William E.,
1300 8th St., Wichita Falls.
Daily, Robert L.,
115 Med. Arts Bldg., Wichita Falls.
Davey, Joseph A., 617 Ave. D., Burkburnett.
Dean, Garland R.,
1500 W. 8th St., Wichita Falls.
Deets, Kenneth H.,
4007 Faith Rd., Wichita Falls.
Donnelly, Allen D., Box 300, Wichita Falls.
Dorbandt, Barton W.,
1411 9th St., Wichita Falls.
Dryden, Charles B., Jr.,
1511 Tenth St., Wichita Falls.
Egdorf, Otto C.,
225 Hamilton Bldg., Wichita Falls.
Eyres, James A. Box 300, Wichita Falls.
Fish, Pascal E., 419 N. Waggoner St., Electra.
Fletcher, Donald E.,
1600 8th St., Wichita Falls.
Fuller, Colonel B., 504 Flood St., Wichita Falls.
Geyer, C. Mac,
102 Med. Arts Bldg., Wichita Falls.
Hall, Joseph D.,
1500 8th St., Wichita Falls.
Hargrave, Robert L.,
218 Hamilton Bldg, Wichita Falls.
Harkins, Thomas A., Box 300, Wichita Falls.
Harrell, Fred S., Olney.
Hathorn, Jerome B., Jr.,
2400 Rushing, Wichita Falls.
Heymann, Julius A.,
1518 10th St., Wichita Falls.
Higgs, Robert E., 1511 10th St., Wichita Falls.
Holt, Joseph G., 1518 10th St., Wichita Falls.
Horany, Melvin, Box 858, Archer City.
Huff, Mark E., 2502 10th St., Wichita Falls.
Humphrey, Irving L., Jr.,
1300 8th St., Wichita Falls.
Humphrey, T. Roger,
2400 Rushing St., Wichita Falls.
Irvine, Eleanor S., 1505 10th St., Wichita Falls.
Irvine, Everett W., Jr.,
1505 10th St., Wichita Falls.
Jackson, John L., III,
1314 9th St., Wichita Falls.
Johnson, James A.,
Hamilton Bldg., Wichita Falls.
Jones, Eldo M., 710 Brook St., Wichita Falls.
Kable, Warren T., Jr.,
1300 8th St., Wichita Falls.
Kanatser, Joseph E.,
1518 10th St., Wichita Falls.
Knight, Ralph W., 710 Brook, Wichita Falls.
Knox, Roland F., 1300 8th St., Wichita Falls.
Krecke, Charles F., (Mil.),
3750th USAF Hosp., Sheppard AFB.
Landon, Fred R., (Hon.),
2410 Ellingham Drive, Wichita Falls.
LeBeau, George L., Jr.,
1518 10th St., Wichita Falls.
Ledbetter, W. Harry,
1518 10th St., Wichita Falls.
Lee, Frank J., 1300 8th St., Wichita Falls.
Lee, James T., 1300 8th St., Wichita Falls.
Ligon, C. Wayne, (Mil.),
4415 McCrory, Wichita Falls.
Little, James A., Staley Bldg., Wichita Falls.
Lovett, James P., Olney.
Lovett, Raymond E., Olney.
Lowry, William P.,
1502 8th St., Wichita Falls.
Lynch, Thomas C., (Hon.),
1704 Austin, Wichita Falls.
Manar, Roger W., 1400 8th St., Wichita Falls.
Mansur, Harl D., Jr.,
1400 8th St., Wichita Falls.
Martinez A, Augustin, Box 300, Wichita Falls.
Maxfield, Jack E., 1300 8th St., Wichita Falls.
McCall, J. Preston,
1300 8th St., Wichita Falls.
McFatridge, Keith W.,
1617 10th St., Wichita Falls.
McKinney, James R.,
2100 Taft St., Wichita Falls.
Meredith, E. Filmore, 306 W. Main, Olney.
Moffitt, O. Peyton, 1300 8th St., Wichita Falls.
Morrison, G. Cameron,
702 Brook, Wichita Falls.
Munro, L. Alton, 1300 8th St., Wichita Falls.
Nail, James B., 1410 8th St., Wichita Falls.
Nail, James B., Jr., 1410 8th St., Wichita Falls.
Nelson, Richard L.,
W. F. Clinic Hosp., Wichita Falls.
Nilasena, Samuel S., 702 Brook, Wichita Falls.
Ogden, William H., (Hon.), Electra.
Owens, Henry B., 1600 8th St., Wichita Falls.
Pace, Robert E., Jr.,
500 Broad St., Wichita Falls.
Parker, William L., 1300 8th St., Wichita Falls.
Parsons, Clyde W., 1300 8th St., Wichita Falls.
Perry, John H., Jr.,
Med. Arts Bldg., Wichita Falls.
Pierce, Alexander W.,
1518 10th St., Wichita Falls.
Pillsbury, Curtis B., 4916 Earl, Wichita Falls.
Powers, Stephen A.,
5th & Broad Sts., Wichita Falls.
Powers, William L.,
1518 10th St., Wichita Falls.
Reagan, John R., 1511 10th St., Wichita Falls.
Rector, William L.,
1518 10th St., Wichita Falls.
Reser, Wayne A., 1504 8th St., Wichita Falls.
Rodriguez, Juan A., (Mil.),
10708 Dunaway Drive, Dallas.
Rosenblatt, William,
1504 8th St., Wichita Falls.
Rosewall, Charles R.,
Box 767, Monterey, Calif.
Scofield, James A., Jr.,
1500 8th St., Wichita Falls.
Seibold, George J.,
702 Brook St., Wichita Falls.
Shaw, Clinton M., Jr.,
1518 10th St., Wichita Falls.
Shepley, Felix R., 1518 10th St., Wichita Falls.
Simmons, Lillard N.,
1518 10th St., Wichita Falls.
Slaughter, George W., III,
1300 8th St., Wichita Falls.
Smith, Percy K.,
W. F. Clinic Hosp., Wichita Falls.
Speakman, Walter F.,
812 Denver St., Wichita Falls.
Steed, Joe D., 1300 8th St., Wichita Falls.
Sullivan, Harley C.,
500 Broad St., Wichita Falls.
Taylor, Fred W., 1511 10th St., Wichita Falls.
Terrell, James Wm.,
4000 Jacksboro Hwy., Wichita Falls.
Terry, Houston H., (Hon.),
3205 Montague, Fort Worth.
Theimer, Louis M.,
115 Med. Arts Bldg., Wichita Falls.
Thompson, John G., Electra.
Thornton, Gail, Jr.,
1502 8th St., Wichita Falls.
Trimble, Orman H.,
110 Med. Arts Bldg., Wichita Falls.
VanDeventer, Loyd R.,
1702 7th St., Wichita Falls.
Walker, William J.,
P. O. Box 300, Wichita Falls.
Watson, Earl F., (Mil.),
2355 Capri Drive, Fort Worth.
Whiting, Walter B.,
1300 8th St., Wichita Falls.
Wilson, Charles H.,
1300 8th St., Wichita Falls.
Wilson, Claude D.,
1300 8th St., Wichita Falls.
Wilson, Oscar W.,
214 Hamilton Bldg., Wichita Falls.
Worthen, John E., Jr.,
Parker Sq. Clinic, Wichita Falls.
Yeager, Ben G.,
Wichita Falls State Hosp., Wichita Falls.
Yero, Oscar, Box 300, Wichita Falls.

WILBARGER

Borchardt, Alvin L., Vernon.
Coleman, William C., Vernon.
Featherston, Elmer W.,
Med. & Surg. Clinic, Vernon.
Hardin, John B., 1912 Pease St., Vernon.
Hollar, Emory D., 1525 Houston St., Vernon.
Lemee, Raymond A., Vernon.
Miller, Bradford W.,
Herring Natl. Bk. Bldg., Vernon.
Mulrhead, James J., Vernon.
Rogers, Albert C., Vernon.
Slaugenhop, James J.,
2805 Wilbarger St., Vernon.
Spaar, Albert P., Vernon.
Steele, Franklin B.,
Herring Bk. Bldg., Vernon.

FOURTEENTH DISTRICT

Dr. R. Mayo Tenery, Waxahachie, Councilor

COLLIN

Allison, Thomas H., Farmersville.
Apple, George W., 1534 K St., Plano.
Boring, Billy Ray, 1911 Westway, McKinney.
Castner, Chas. W., Rusk State Hosp., Rusk.
Eller, Thomas O., 1305 16th St., Plano.
Erwin, J. C., Jr.,
120½ W. Virginia, McKinney.
Hill, Mack M., Jr.,
317 N. Tennessee, McKinney.
Hooper, John M.,
120½ W. Virginia, McKinney.
Kirby, Vernon V., Jr., Box 387, Roscoe.
Linstrum, Tom E.,
Graves & Harroun, McKinney.
McKellar, Duncan L.,
303 W. Virginia, McKinney.
Mitchell, Glenn C., Box 457, McKinney.
Pink, Erwin G., Frisco.
Quattlebaum, James E.,
311 N. Tennessee, McKinney.
Searcy, Marshall M.,
Graves & Harroun, McKinney.
Shumway, Charles M.,
407 S. Tennessee, McKinney.
Trimble, Terrill M., Wylie Clinic, Wylie.
Truett, Harvey K.,
403 S. Tennessee, McKinney.
Webb, Jack L., Box 192, Farmersville.
Wysong, Charley E., Wysong Clinic, McKinney.
Wysong, H. Dudley, Wysong Clinic, McKinney.
Wysong, W. Scott, Jr.,
Wysong Clinic, McKinney.

COOKE

Alston, Herbert M.,
502 E. Broadway, Gainesville.
Atchison, James W.,
215 S. Commerce, Gainesville.
Brown, Dan M., 110 E. Pecan St., Gainesville.
Davenport, Howard S., 318 E. Elm, Gainesville.
Kaden, Van G., 206 Schad & Pulte, Gainesville.
Lively, Gerald A., Box 296, Whitesboro.
Lusk, Clu Flu, Box 736, Gainesville.
McLeroy, Robert L., 528 S. Dixon, Gainesville.
Mills, Charles K., Box 414, Gainesville.
Powell, William P., Box 721, Gainesville.
Saikin, Stanley E., 110 E. Pecan, Gainesville.
Shea, John D., Box 532, Gainesville.
Thomas, Ira L., 215 S. Commerce, Gainesville.

DALLAS

Abrams, Raymond H.,
3707 Gaston Ave., Dallas.
Adam, Maurice, 3707 Gaston Ave., Dallas.
Adams, Reuben H., S.W. Med. School, Dallas.
Addison, Jack J., 6053 Sherry Lane, Dallas.
Adin, Louis E., Jr., 3707 Gaston, Dallas.
Adwan, Kenneth O.,
10023 Garland Rd., Dallas.
Albright, James E., (Int.),
Baylor Hospital, Dallas.
Aldredge, Geo. N., Jr.,
623 Med. Arts. Bldg., Dallas.
Alexander, Jo C., 3707 Gaston, Dallas.
Alexander, Lee J., Med. Arts Bldg., Dallas.
Alexander, Sam A., 3707 Gaston, Dallas.
Alfieri, Anthony L.,
3427 Cedar Springs, Dallas.
Allday, Louie E., 2929 Welborn, Dallas.
Allen, John B., 4105 Live Oak, Dallas.
Allen, S. Thomas, (Mil.),
Santa Clara Co. Hosp., San Jose, Calif.
Allison, Joe M., Box 215, Grapevine.
Allman, Robert M., Jr., (Mil.),
U. S. P. H. S. Hosp., Seattle, Wash.
Almand, James R., Jr.,
305 N.W. Third St., Grand Prairie.
Altick, Frank J.,
9528 Webbs Chapel Rd., Dallas.
Altman, William A., 202 W. 10th St., Dallas.
Andreassian, M. G., 8215 Westchester, Dallas.
Anthony, James M., 8215 Westchester, Dallas.
April, Paul A., (Int.),
Squibb Inst. for Med. Res., New York, N. Y.
Archer, Richard K., (Int.),
10125 Brockbank, Dallas.
Armstrong, J. Otis, 3810 Swiss Ave., Dallas.
Arnold, Lawrence E., 3607 Gaston, Dallas.
Arnspiger, Larry A., 1511 N. Beckley, Dallas.
Aronoff, Billie L., 712 N. Washington, Dallas.
Aronson, Howard S.,
926 Med. Arts Bldg., Dallas.
Ashby, John E., 3610 Fairmount, Dallas.
Ashe, William M., 712 N. Washington, Dallas.
Ashworth, Charles T.,
5323 Harry Hines, Dallas.

Aten, Eugene L., 8215 Westchester, Dallas.
Atkinson, Geo. N., Jr.,
101 N. Zangs Blvd., Dallas.
Austin, Dale J., 1008 N. Washington, Dallas.
Austin, Frank H., 8215 Westchester, Dallas.
Austin, Ona Mae, 101 S. Iowa, Irving.
Bagwell, John S., 3710 Swiss, Dallas.
Bailey, Herbert A.,
1414 Med. Arts Bldg., Dallas.
Baird, Sydney S., Box 28, Dallas.
Baird, William D., 10915 Garland Rd., Dallas.
Baker, B. Orland, Jr., (Mil.),
Wright-Patterson AFB, Dayton, Ohio.
Baker, John O., 111 E. Woodin, Dallas.
Bakker, Johannes P.,
424 Lochwood Village, Dallas.
Baldwin, Alvin, Jr., 3707 Gaston, Dallas.
Baldwin, James L.,
1627 Med. Arts Bldg., Dallas.
Baldwin, Marion C. L., (Int.),
5916 Azalea Lane, Dallas.
Baldwin, Richard M., (Int.),
Parkland Mem. Hosp., Dallas.
Ball, James A., 10801 Garland Rd., Dallas.
Balla, George A., 3707 Gaston Ave., Dallas.
Barekman, Wm. H., 3607 Gaston Ave., Dallas.
Barkman, Floyd J., 1603 Junior Dr., Dallas.
Barnes, Dorsey K., 3701 Maple, Dallas.
Barnes, John W., (Int.),
St. Pauls Hosp., Dallas.
Barnes, Thomas S., 118 S. Edgefield, Dallas.
Barnett, Jack A., (Int.),
Parkland Mem. Hosp., Dallas.
Barnett, William D., 3707 Gaston Ave., Dallas.
Barr, W. Tom, Medical Arts Bldg., Dallas.
Barron, Stewart S., 4020 Junius St., Dallas.
Barrow, Rex A., Jr., 2601 Welborn, Dallas.
Barton, Robert M., 3601 Swiss Ave., Dallas.
Barzune, Benjamin, 1301 Turtle Creek, Dallas.
Bashour, Samuel B.,
2020 W. Grauwyler, Irving.
Baskin, John L., 8215 Westchester, Dallas.
Bass, James W., 1936 Amelia, Dallas.
Bass, Robert K., 3330 Douglas St., Dallas.
Bassett, Wallace H., 3707 Gaston, Dallas.
Bates, Richard D., 109 S. Delaware, Irving.
Baum, John, (Int.), 5323 Harry Hines, Dallas.
Bayoud, George S.,
Miller at Shiloh Rd., Garland.
Bayoud, Paige B., 9323 Garland Rd., Dallas.
Beall, John R., 2615 Cole Ave., Dallas.
Beaver, Norman B., (Hon.),
4th U. S. Army, Ft. Sam Houston.
Beck, George P., Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Beck, Jay M., (Int.),
1360th USAF Hosp., Orlando, Fla.
Beckering, Henry H., 3701 Maple, Dallas.
Bell, Marvin D.,
1109 Med. Arts Bldg., Dallas.
Bell, Wm. Sterling, 3505 Turtle Creek, Dallas.
Bennett, Katharine P., 915 St. Joseph, Dallas.
Bennett, Thomas R., Jr.,
3707 Gaston Ave., Dallas.
Berbary, Maurice S., (Mil.),
A. F. Inst. of Path., Washington 25, D.C.
Berk, William R., 1909 Second Ave., Dallas.
Bernhardt, Donald R., Med. Arts Bldg., Dallas.
Best, Abraham R., 101 S. Iowa, Irving.
Biggart, James F., Jr., 3607 Gaston, Dallas.
Binion, John T., 3707 Gaston Ave., Dallas.
Bishop, Robert T. E.,
3988 N. Central Expressway, Dallas.
Bisland, Theodore, 3707 Gaston Ave., Dallas.
Blain, James M., Baylor Hosp., Dallas.
Blair, D. Shelton,
1609 Med. Arts Bldg., Dallas.
Blanton, Bassel N., 532 Med. Arts Bldg., Dallas.
Blend, Max H., 4208 Swiss Ave., Dallas.
Block, Harold M., Med. Arts Bldg., Dallas.
Block, Richard E., 712 N. Washington, Dallas.
Bloss, Charles L.,
1420 Med. Arts Bldg., Dallas.
Boehning, Harold C., 4206 Swiss Ave., Dallas.
Boland, Grant L., 712 N. Washington, Dallas.
Bondy, Robert C., Jr.,
203 W. Tyler, Richardson.
Bone, Fred W., 3702 Worth, Dallas.
Bonte, Frederick J., 5201 Harry Hines, Dallas.
Bookatz, Allan, 3707 Gaston, Dallas.
Boone, Clifford M., 121 S. Zangs, Dallas.
Boone, Thetford B., Jr.,
1315 Stemmons, Dallas.
Booth, James, 3330 S. Lancaster, Dallas.
Bornstein, David M., 3707 Gaston Ave., Dallas.
Boswell, George M., Jr.,
364 Casa Linda Pl., Dallas.
Bounds, Murphy, Medical Arts Bldg., Dallas.
Bourland, J. W., Jr., 4105 Live Oak, Dallas.
Bourland, J. W., Sr., (dead), Dallas.
Bourland, John B., Box 28, Dallas.
Bowden, Ben W., 10801 Garland Rd., Dallas.
Boyer, Lorenzo A., 7117 Mil. Parkway, Dallas.
Bracken, Frank L.,
111 N.E. 11th St., Grand Prairie.
Bradfield, James Y., 3601 Swiss Ave., Dallas.
Bradfield, John L., Med. Arts Bldg., Dallas.
Bradford, Harry M., (Hon.),
1232 N. Selva Dr., Dallas.
Bralley, Ernest M., Jr., (Mil.),
Dewitt Army Hosp., Ft. Belvoir, Va.
Branch, George R., 4143 Cole Ave., Dallas.
Branch, William M.,
712 N. Washington, Dallas.
Brannin, Dan, 929 Med. Arts Bldg., Dallas.
Brau, J. Gilmore, 3817 Brown St., Dallas.
Breihan, E. W., Box 5297, Dallas.
Brereton, Gilbert E.,
723 Medical Arts Bldg., Dallas.
Brooking, Donald G. W.,
414 Medical Arts Bldg., Dallas.
Brooks, Ernest J., (Hon.),
1312 N. Henry Rd., Arlington.
Brooks, William C., (Int.),
St. Pauls Hosp., Dallas.
Brooksaler, Fred S., 8215 Westchester, Dallas.
Browder, Maurice M.,
2024 W. Grauwyler, Irving.
Brown, C. Frank, P. O. Box 2699, Dallas.
Brown, Donald S., 3710 Swiss, Dallas.
Brown, Harold, 1400 Marion, Garland.
Brown, Olen E., (Hon.),
8903 S. Beckley, Dallas.
Brown, Samuel R., 219 W. 10th St., Dallas.
Browning, Carroll W., 101 N. Zangs, Dallas.
Bruce, Charles H.,
1317 N. Washington, Dallas.
Bruce, R. Grady, 209 Med. Arts Bldg., Dallas.
Brunken, Robert B., 3707 Gaston Ave., Dallas.
Bruton, Emmett B., 4217 Swiss Ave., Dallas.
Bryan, Nelson A., (Hon.),
978 Peavy Rd., Dallas.
Bryan, Robert T., Jr., 3511 Fairmount, Dallas.
Bryant, Richard E., (Int.),
Parkland Mem. Hosp., Dallas.
Buchanan, Ronald E., (Int.),
St. Paul Hosp., Dallas.
Buckholts, Walter H., V.A. Hospital, Dallas.
Buehler, Martin S., 3225 Turtle Creek, Dallas.
Buell, David S., 8215 Westchester, Dallas.
Buford, Ben R., 1425 Med. Arts Bldg., Dallas.
Buford, Lee Owen S., 2514 Welborn St., Dallas.
Bullion, Charles F.,
233 W. Wheatland Rd., Dallas.
Bumpass, S. R., 915 St. Joseph, Dallas.
Burford, Raymond W., 4105 Live Oak, Dallas.
Burkett, Howard M.,
1530 Med. Arts Bldg., Dallas.
Burnett, Jack F., (Mil.,) Dallas.
Burns, Maudie Marie, Med. Arts Bldg., Dallas.
Burnside, Ronald M., 4105 Live Oak, Dallas.
Bush, Oliver F., 712 N. Washington, Dallas.
Bush, W. Leslie, 3707 Gaston, Dallas.
Bussey, C. D., Med. Arts Bldg., Dallas.
Butte, Felix L.,
1224 Med. Arts Bldg., Dallas.
Byers, Jerome L.,
206 Wynnewood Prof. Bldg., Dallas.
Byrom, Emmett T.,
1603 Medical Arts Bldg., Dallas.
Bywaters, T. W., 3607 Gaston, Dallas.
Caillet, O. Rene, 8215 Westchester, Dallas.
Cairns, A. B., 3511 Fairmount, Dallas.
Caldwell, Joe B., 4105 Live Oak St., Dallas.
Calhoun, Nina Fay,
1532 Medical Arts Bldg., Dallas.
Cameron, Lawrence C., 4407 Oak Lawn, Dallas.
Camp, Thomas F., Jr., (Int.),
48th T.A.C. Hosp., New York, N. Y.
Campbell, Allen D., Jr.,
1202 N. Beckley, Dallas.
Campbell, Donovan, 4206 Swiss Ave., Dallas.
Campbell, Robert D.,
10023 Garland Rd., Dallas.
Campbell, Robert G., 8215 Westchester, Dallas.
Canter, Dennis, 4224 Swiss Ave., Dallas.
Carlson, Glenn D.,
1221 Med. Arts Bldg., Dallas.
Carman, H. Frank,
830 Med. Arts Bldg., Dallas.
Carney, James P., 1218 W. Jefferson, Dallas.
Carpenter, Robert G., 1203 N. Madison, Dallas.
Carrell, Brandon, 3701 Maple, Dallas.
Carroll, Benjamin H., 2600 Welborn, Dallas.
Carswell, Winston E.,
Medical Arts Bldg., Dallas.
Carter, C. B., Med. Arts Bldg., Dallas.
Carter, David W., Jr.,
3707 Gaston Ave., Dallas.
Carter, Earl L., 528 Med. Arts Bldg., Dallas.
Carter, H. Gray, 712 N. Washington, Dallas.
Carter, Norman W.,
S.W. Medical School, Dallas.
Chandler, H. Norman, 6331 Prospect, Dallas.
Chaney, Clyde E., 10346 Ferguson Rd., Dallas.
Chapman, Carleton B.,
5323 Harry Hines, Dallas.
Chapman, John S., 5323 Harry Hines, Dallas.
Chears, W. Crockett, Jr.,
1104 Med. Arts Bldg., Dallas.
Cheek, J. Harold, 3707 Gaston Ave., Dallas.
Cherry, Glenn R.,
909 Med. Arts Bldg., Dallas.
Chester, Catherine P., 3330 S. Lancaster, Dallas.
Chester, John B., 3330 S. Lancaster, Dallas.
Chisolm, Jack T.,
1606 Med. Arts Bldg., Dallas.
Christianson, Lynn C.,
3819 Maple Ave., Dallas.
Cirone, Vincent C., (Int.),
1016 N. Mont Clair, Dallas.
Clariday, John M., 9528 Webbs Chapel, Dallas.
Clark, Arthur L., Med. Arts Bldg., Dallas.
Clark, Fannie M., 3829 Hall St., Dallas.
Clark, Harold G., Med. Arts Bldg., Dallas.
Clark, Helen, 10317 Hedgeway Dr., Dallas.
Clark, William K., 5323 Harry Hines, Dallas.
Clarkson, Alwin M., 526 E. Jefferson, Dallas.
Cleveland, Edwin M., 4105 Live Oak, Dallas.
Cobb, Joseph B., 9323 Garland Rd., Dallas.
Cobb, Stephen W., 317 N. Zangs, Dallas.
Coggeshall, Howard C., 3710 Swiss, Dallas.
Cole, Charles Max,
1421 Med. Arts Bldg., Dallas.
Cole, Gillon M., 1315 Eastus Dr., Dallas.
Colip, William L.,
330 N.W. 2nd St., Grand Prairie.
Collier, Gates, (Inac.), 511 N. Akard, Dallas.
Collier, Richard E., Baylor Hosp., Dallas.
Combes, Burton, 5323 Harry Hines, Dallas.
Connor, Robert B., 8215 Westchester, Dallas.
Conrad, Emmett J., 4303 Oakland, Dallas.
Conyers, Loyd W.,
906 Med. Arts Bldg., Dallas.
Cook, Chester E., Box 2699, Dallas.
Cooper, Jack C., (Mil.),
902 Med. Arts Bldg., Dallas.
Copeland, Horace V., (Hon.),
125 S.W. Dallas St., Grand Prairie.
Copps, James M., 4105 Live Oak St., Dallas.
Corgill, Donald A., V.A. Hospital, Dallas.
Coufal, Frank H., Jr., (Int.),
Baylor Hospital, Dallas.
Courtin, Raymond F., 4224 Swiss Ave., Dallas.
Cox, Eli R., 6331 Prospect, Dallas.
Cox, Kelly, 630 Med. Arts Bldg., Dallas.
Cox, Virgil M., Jr., (Int.),
Duke Hosp., Durham, N. C.
Crane, Frances B., 238 W. 10th, Dallas.
Crane, William D., 238 W. 10th, Dallas.
Crass, Gwendolyn, 712 N. Washington, Dallas.
Crawford, Franklin L.,
501 Jefferson St., Cedar Hill.
Crenshaw, Allen, Jr., 3707 Gaston, Dallas.
Crow, William E.,
1028 Medical Arts Bldg., Dallas.
Crutcher, Howard K., 1511 N. Beckley, Dallas.
Crutcher, Howard K., Jr., (Int.),
2929 Milton, Dallas.
Culbertson, Marvin C.,
3619 Fairmount, Dallas.
Culmer, Harold H., 2626 Bryan, Dallas.
Culwell, Jerry H., 642 E. 2nd St., Irving.
Cupp, Charles D.,
1114 E. Main, Grand Prairie.
Curtess, Ross L., Jr., 3607 Gaston Ave., Dallas.
Cushing, Ivan E., Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Daily, William M., 3707 Gaston Ave., Dallas.
Daniel, Ruby K.,
706 Med. Arts Bldg., Dallas.
Darrough, Lawrence E., 4105 Live Oak, Dallas.
Dathe, Richard A., 3707 Gaston, Dallas.
Daughety, Jewel S., (Int.),
Baylor Med. Center, Dallas.
Davidson, G. A., 4105 Live Oak, Dallas.
Davidson, Joseph W., Jr.,
Methodist Hospital, Dallas.
Davidson, Vanda A.,
712 N. Washington, Dallas.
Davis, Leo G., 521 Med. Arts Bldg., Dallas.
Davis, Milton V., 3707 Gaston Ave., Dallas.
Dawson, J. L., (Hon.),
4535 S. Versailles, Dallas.
Dean, William B., 238 W. 10th St., Dallas.
DeBolt, Merlan E., 5311 Farquhar Lane, Dallas.
deHaro, J. Anthony, 2923 Fairmount, Dallas.
DeLange, Arnott, 1511 N. Beckley, Dallas.
deLarios, Arthur T., 3631 Fairmount, Dallas.
Denman, John L., 712 N. Washington, Dallas.
Denny, Ben W., 1511 N. Beckley, Dallas.
Denton, Guy T., Jr.,
1311 N. Stemmons, Dallas.
Denton, Guy T., Sr., (Hon.),
1002 N. Edgefield, Dallas.
D'Errico, Albert P., 3707 Gaston, Dallas.
Devereux, Wm. P.,
1311 N. Washington, Dallas.
Dewlett, Hal J.,
8107 Chadbourne Rd., Dallas.
Dierolf, Leon W., Jr.,
1511 N. Beckley, Dallas.
Dimmette, James E., 3607 Gaston Ave., Dallas.

Disch, Ralph C., 3330 S. Lancaster, Dallas.
Dixon, Ward G.,
1420 Med. Arts Bldg., Dallas.
Dixon, Warren F.,
311 Preston Royal Med. Cen., Dallas.
Dombrowsky, Edward F.,
233 W. 10th St., Dallas.
Donoho, Charles P., 3707 Gaston Ave., Dallas.
Dorman, George W., 3524 Fairmount, Dallas.
Douglas, F. Clark,
1414 Med. Arts Bldg., Dallas.
Downs, James T., Jr.,
8540 San Fernando, Dallas.
Downs, James T., III, 3707 Gaston, Dallas.
Driggs, Guy K.,
724 Town Creek Dr., Dallas.
Duckett, J. Warner,
1004 N. Washington, Dallas.
Duncan, Charles N., 6003 Victor, Dallas.
Duncan, Horace E., 4500 Lancaster, Dallas.
Dunlap, Elbert, (Hon.),
5339 Nakoma, Dallas.
Dunlap, J. Hudson, 4000 Junius, Dallas.
Dunn, Fred H., (Int.),
Michael Reese Hosp., Chicago, Ill.
Dykes, Thomas L., 1121 W. Jefferson, Dallas.
Edwards, J. Bruce, 4342 Lovers Lane, Dallas.
Edwards, W. L. Jack, 3710 Swiss, Dallas.
Eisenberg, Alex A., 3707 Gaston Ave., Dallas.
Elder, Lindsey T., Jr.,
1511 N. Beckley, Dallas.
Emmert, John A., 8215 Westchester, Dallas.
Emmett, George Earl,
712 N. Washington, Dallas.
Ensey, James E.,
310 Preston Royal Med. Cen., Dallas.
Epstein, Maurice W., (Mil.),
814th Hosp. Group, Westover AFB, Mass.
Ericson, Ruth Ann., 4227 Herschel, Dallas.
Ernst, Richard W., 5323 Harry Hines, Dallas.
Erwin, Karl W., 3531 Fairmount, Dallas.
Estes, Ivan A., 8220 Westchester, Dallas.
Evans, Allan C., (Mil.),
304 Shreveport Rd., Barksdale AFB, La.
Evans, Edward L.,
1038 E. Kessler Pkwy., Dallas.
Evans, Walter G., (Hon.),
4065 Purdue, Dallas.
Fader, David E., 3707 Gaston, Dallas.
Fain, Allen M., Duncanville.
Fain, Guy Burton, (Hon.),
2431 Catherine, Dallas.
Falke, Clarence G.,
Wynnewood Prof. Bldg., Dallas.
Fallis, Bruce D., 5323 Harry Hines, Dallas.
Fangman, Donald F.,
110E Walnut Hill Village, Dallas.
Fanoni, Roy H., (Int.), 101 S. Iowa, Irving.
Fashena, Gladys J., S.W. Med School, Dallas.
Feldman, Jerald, (Int.),
Parkland Mem. Hosp., Dallas.
Ferguson, Doyle W., 3631 Fairmount, Dallas.
Fiegel, Walter L., Box 576, Carrollton.
Fine, Jacob S., 3614 Fairmount, Dallas.
Fine, Myron G., 616 Med. Arts Bldg., Dallas.
Fink, Frederick,
13500 N. Central Expressway, Dallas.
Finley, Dorothy G.,
156 Wynnewood Prof. Bldg., Dallas.
Finnegan, Chas. R.,
610 Med. Arts Bldg., Dallas.
Fisch, Ben R., (Int.),
816 Morreene Rd., Durham, N. C.
Fischer, J. Philip, 3601 Swiss Ave., Dallas.
Fish, Stewart A.,
1311 N. Washington, Dallas.
Fisher, John W.,
1414 Med. Arts Bldg., Dallas.
Fisher, Thomas B., (Hon.),
3620 Armstrong, Dallas.
Flohr, Leonard J.,
Medical Arts Bldg., Dallas.
Flowers, W. K., Jr., 2317½ Hall St., Dallas.
Fogelman, Morris J.,
712 N. Washington, Dallas.
Fogleman, James D.,
535 W. Pleasant Run Rd., Lancaster.
Foree, Kenneth, III,
631 Med. Arts Bldg., Dallas.
Foster, Fred M., (Int.),
1315 Stemmons, Dallas.
Fowler, Hanes M.,
Wynnewood Prof. Bldg., Dallas.
Fox, Everett C., Med. Arts Bldg., Dallas.
Frank, Gordon D., 3707 Gaston Ave., Dallas.
Fromm, Chas. S., 3707 Gaston, Dallas.
Fuller, William W., 6200 Lemmon, Dallas.
Funk, Presley C., 9528 Lemmon Ave., Dallas.
Fuqua, Foster, 3707 Gaston, Dallas.
Fuqua, W. N., Jr., 3707 Gaston, Dallas.
Gaines, Sidney W.,
134 E. Church, Grand Prairie.
Galt, Jabez, 510 Med. Arts Bldg., Dallas.
Galt, Sidney, 220 Med. Arts Bldg., Dallas.
Garfiss, Francisco G.,
5535 Denton Dr., Dallas.
Garner, Amos D., 6331 Prospect, Dallas.
Garrett, Richard E., (Int.),
Parkland Mem. Hosp., Dallas.
Garvey, Ronald F., (Int.),
5323 Harry Hines, Dallas.
Geary, Francis B., Jr., 219 S. Willomet, Dallas.
Geibel, John Wm., Jr., 4105 Live Oak, Dallas.
Geist, Frederick S.,
Wynnewood Prof. Bldg., Dallas.
Gerard, Marvin, (Mil.),
USAF Hosp., Carswell AFB.
Gibbons, O. W., Med. Arts Bldg., Dallas.
Gibbons, Raymond R., (Int.),
Parkland Mem. Hosp., Dallas.
Gibbons, Wm. E., Med. Arts Bldg., Dallas.
Gilbert, Francis M., 101 S. Iowa, Irving.
Gilbert, Franklin M., 101 S. Iowa, Irving.
Gilbert, Stanley L.,
9528 Webbs Chapel Rd., Dallas.
Gilbert, Taylor C.,
1316 Med. Arts Bldg., Dallas.
Giles, Robert B.,
1114 Med. Arts Bldg., Dallas.
Giles, Robert B., Jr.,
1114 Med. Arts Bldg., Dallas.
Gill, Atticus J., 7103 Lakewood Blvd., Dallas.
Gill, Dan C., 3707 Gaston, Dallas.
Gilmore, Jack T., 9429 El Centro Dr., Dallas.
Ginderske, Monica M.,
10915 Garland Rd., Dallas.
Girard, Percy M., 2601 Welborn, Dallas.
Gleichert, James E., 1511 N. Beckley, Dallas.
Godfrey, William A., Jr., (Int.),
V. A. Hospital, Dallas.
Goff, Gomer F., (dead), Dallas.
Goforth, John L., Box 1074, Dallas.
Goode, James E., 737 N. Bishop, Dallas.
Goode, John V., 3707 Gaston Ave., Dallas.
Goodfried, Max Paul, 3814 Swiss, Dallas.
Goodlad, James K.,
1006 W. Camp Wisdom, Dallas.
Goodson, James H.,
10801 Garland Rd., Dallas.
Gordon, Joseph B., 3707 Gaston Ave., Dallas.
Gorman, James A., Jr., (Mil.),
1360th USAF Hosp., Orlando AFB, Fla.
Gossard, Wayne H., 135 Melba, Dallas.
Goth, Andres, 5323 Harry Hines, Dallas.
Gottlich, Arthur F.,
800 N. Washington, Dallas.
Grace, Robert W., 6331 Prospect, Dallas.
Grafton, Edwin G., 8215 Westchester, Dallas.
Graham, James F., 1418 W. Jefferson, Dallas.
Graham, Marion F., 9323 Garland Rd., Dallas.
Graham, Russell B., 4105 Live Oak, Dallas.
Gran, Gerson, 3427 Cedar Springs, Dallas.
Grater, William C.,
1620 Med. Arts Bldg., Dallas.
Graves, Ghent, Jr., (Int.), V.A. Hosp., Dallas.
Gray, George A., 3700 Ross Ave., Dallas.
Gray, Jack A., (Mil.),
U. S. Army Hosp., Fort Hood.
Green, Adam D., 4105 Live Oak, Dallas.
Green, F. Ray, 5511 Hudson, Dallas.
Green, Tim R., 1107 W. Jefferson, Dallas.
Green, William, 3205½ Thomas Ave., Dallas.
Green, William T., 6003 Victor, Dallas.
Green, Willie Joe, 5535 Denton Dr., Dallas.
Gregory, Charles F., 5323 Harry Hines, Dallas.
Greve, Marion J., 3707 Gaston, Dallas.
Griffin, Jack B., 800 N. Washington, Dallas.
Griffith, E. Clay, (Int.),
P. O. Box 1769, Dallas.
Grigson, James P., Jr., (Int.),
Baylor Med. Center, Dallas.
Grollman, Arthur, 5323 Harry Hines, Dallas.
Gross, Norman H., 3614 Fairmount, Dallas.
Gross, Perry E., 3707 Gaston Ave., Dallas.
Grossman, Mervin H., P. O. Box 57, Dallas.
Grover, Frederick W., (Int.),
Childrens Med. Cen., Dallas.
Grow, Max H., 3616 Maple Ave., Dallas.
Guerriero, Wm. F.,
712 N. Washington, Dallas.
Guynes, Henry C.,
10915 Garland Rd., Dallas.
Hackney, U. P., (Hon.),
Medical Arts Bldg., Dallas.
Hale, Martha Helen, 3403 Hall, Dallas.
Halebian, Hratch S.,
806 Med. Arts Bldg., Dallas.
Haley, Arvel E., 737 N. Bishop, Dallas.
Haley, William E.,
702 Med. Arts Bldg., Dallas.
Hall, William L., (Inac.),
5435 Singing Hills, Dallas.
Halley, B. C., Jr., 3707 Gaston, Dallas.
Halpern, Salmon R., 3534 Maple, Dallas.
Hamilton, Chas. F.,
1616 Med. Arts Bldg., Dallas.
Hancock, Warren E., 9323 Garland Rd., Dallas.
Hare, Henry P., Jr.,
210 N. Westmoreland, Dallas.
Harper, Jack C., 526 E. Jefferson, Dallas.
Harrel, Don G., 220 W. Oakenwald, Dallas.
Harrell, Andrew J., (Int.),
Methodist Hosp., Dallas.
Harrell, Ronald R.,
108 N.E. 7th St., Grand Prairie.
Harrington, Francis T.,
9962 Rock Brook Lane, Dallas.
Harrington, Marion Ray,
8215 Westchester, Dallas.
Harrington, S. F., 8215 Westchester, Dallas.
Harris, Alfred W., 3710 Swiss, Dallas.
Harris, Calvin W., 219 W. 10th St., Dallas.
Harris, N. Joe, 8215 Westchester, Dallas.
Harris, Oscar T., Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Harris, William S., 4020 Junius, Dallas.
Harris, Worth W., 400 S. Ebrite, Mesquite.
Harrison, Ben F., Jr., 3707 Gaston, Dallas.
Harrison, Edwin E., Jr., (Int.),
Baylor Med. Center, Dallas.
Harrison, Frank, S.W. Med. School, Dallas.
Harrison, Gaston G., 6115 La Vista, Dallas.
Hart, Gaylord A., Med. Arts Bldg., Dallas.
Hartin, Richard B., 3110 Lamesa, Garland.
Hartman, James M., 819 Ave. A, Garland.
Harvill, T. Haynes,
1616 Med. Arts Bldg., Dallas.
Hawkins, Hubert F., 3607 Gaston, Dallas.
Hawkins, Wm. C., 4143 Cole Ave., Dallas.
Hayes, Elmer R., 3707 Gaston, Dallas.
Hayes, Horace T., 289 Casa Linda Pl., Dallas.
Haynes, William M.,
Wynnewood M. & P. Bldg., Dallas.
Healy, Michael J., 1203 Gordon, Dallas.
Hellman, Wayne L., (Mil.),
98 Gen. Hosp., APO 34, New York, N.Y.
Hempel, G. Kennedy, Jr., (Int.),
3626 Granada, Dallas.
Henderson, Clyde D., Jr.,
306 Tyler, Richardson.
Henderson, Hodgie C., Jr.,
220 W. Oakenwald, Dallas.
Hendler, Albert F., 9323 Garland Rd., Dallas.
Henner, Charles M., Methodist Hosp., Dallas.
Henniger, Waldemar F., Jr.,
10915 Garland Rd., Dallas.
Henry, Albert C., Jr., 3707 Gaston Ave., Dallas.
Henry, David J., 3607 Gaston Ave., Dallas.
Henry, James W.,
2539 S. Garland Rd., Garland.
Henry, Joseph M.,
205 Marshall Lane Shopping Village, Dallas.
Hernandez, Onesimo, 3531 Fairmount, Dallas.
Herndon, Ernest T., 8215 Westchester, Dallas.
Herndon, James H.,
510 Med. Arts Bldg., Dallas.
Herrick, Richard B.,
531 Med. Arts Bldg., Dallas.
Hershey, Edythe P., 1114 Commerce St., Dallas.
Hess, Evelyn V., (Int.),
5323 Harry Hines, Dallas.
Hesser, Robert N., 3614 Fairmount, Dallas.
Heyer, Howard E., 3601 Swiss Ave., Dallas.
Hibbitts, William M.,
1631 Med. Arts Bldg., Dallas.
Higginbotham, Juanita, (Int.),
Timberlawn San, Dallas.
Hill, James R., 2339 Gus Thomasson, Dallas.
Hill, Joseph M., 6752 Avalon, Dallas.
Hill, S. M., Med. Arts Bldg., Dallas.
Hinnant, Jerry H., (Int.),
V. A. Hospital, Dallas.
Hodges, F. Carlton, Jr., (Int.),
Baylor Med. Center, Dallas.
Hodges, Harold C., 1508 S. Buckner, Dallas.
Hodges, Leon, 4407 Oak Lawn, Dallas.
Hodges, T. Wiley, 9323 Garland Rd., Dallas.
Hoefer, Carl A., (Inac.),
1379 Hill Dr., Los Angeles, Calif.
Hoekstra, Clarence S.,
8215 Westchester, Dallas.
Hoffman, Glenden A., Med. Arts Bldg, Dallas.
Hoffman, Mary S.,
7431 Midbury Drive, Dallas.
Hoffman, William W.,
4105 Live Oak St., Dallas.
Holland, John, III, 4224 Swiss Ave., Dallas.
Holman, James, Med. Arts Bldg., Dallas.
Holt, C. Zeno, 115 W. Preston For., Dallas.
Holt, J. O. S., Jr., 3707 Gaston Ave., Dallas.
Hood, Marianna, 3707 Gaston, Dallas.
Hopkins, May Agness,
1032 Med. Arts Bldg., Dallas.
Horgan, John D.,
117 Kiest & Polk Village, Dallas.
Horn, Fred W., 5535 Denton Dr., Dallas.
Horn, J. Morris, 3707 Gaston, Dallas.
Hough, Jerald P., 1511 N. Beckley, Dallas.
Houser, Edward F., Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Howard, Ben K., 712 N. Washington, Dallas.
Howard, Charles H., Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Howard, George W., 628 Haines, Dallas.

Howell, Benjamin E.,
3016½ Thomas Ave., Dallas.
Howell, James B., Med. Arts Bldg., Dallas.
Hubbard, David G., 3525 Turtle Creek, Dallas.
Hubbard, Wm. F., Jr., (Int.),
St. Paul Hosp., Dallas.
Hudson, W. Lee, (Hon.),
4312 Overhill Drive, Dallas.
Huggins, Clifford S., (Int.),
V. A. Hospital, Dallas.
Hunsinger, J. Edward,
3988 Central Exp., Dallas.
Hunt, Samuel Lee, 10023 Garland Rd., Dallas.
Hunter, Richard B., 7229 Lehigh, Dallas.
Hurt, George E., Jr., (Int.),
Parkland Hosp., Dallas.
Hurt, Leonard B., 3917 Miramar, Dallas.
Hutton, Sam B., Jr., 3707 Gaston, Dallas.
Iliya, Adil, 10657 Lakemere, Dallas.
Irving, W. M., Jr., 820 Irving Blvd., Irving.
Jackson, Claude L., Box 1769, Dallas.
Jackson, Jimmy D., 5511 Hudson, Dallas.
Jackson, Mary Ruth, 3629 Fairmount, Dallas.
Jackson, Michael C., 6315 Prospect, Dallas.
Jacobson, Coleman, 3707 Gaston, Dallas.
James, George Taylor, 3631 Fairmount, Dallas.
James, George Truett, 3701 Maple, Dallas.
James, John A., Southwest Med. Schl., Dallas.
Jamison, Harwin B., 1511 N. Beckley, Dallas.
Jenkins, John L., 722 Med. Arts Bldg., Dallas.
Jenkins, Marion T., Parkland Hosp., Dallas.
Jenkins, Speight, 814 Med. Arts Bldg., Dallas.
Jennings, Mary A., 4210 Lemmon, Dallas.
Jerome, Johnnie Jeff, (Mil.),
USA Disp. APO 503, San Francisco, Calif.
Johnson, Alvis F., Jr., 2306 Welborn, Dallas.
Johnson, William B., 6331 Prospect, Dallas.
Johnston, Louis C., 10th Flr. Med. Arts., Dallas.
Jones, George M., 3601 Swiss Ave., Dallas.
Jones, Homer C., Jr.,
2317 Mountain Lake Rd., Dallas.
Jones, J. Guy, 2600 Welborn, Dallas.
Jones, J. William, 220 Med. Arts Bldg., Dallas.
Jones, Robert A., Box 398, Lancaster.
Jones, Wendell A., (Int.),
Baylor Med. Center, Dallas.
Jones, William D., 3116 Live Oak, Dallas.
Jordan, Frank H., 2918 Thomas Ave., Dallas.
Jordan, Fred H., 210 N. Westmoreland, Dallas.
Jordan, J. Russell, 3607 Gaston, Dallas.
Jordan, Lois F., 3607 Gaston, Dallas.
Kadesky, Melvin C., 3707 Gaston Ave., Dallas.
Kahler, E. Clayton, Med. Arts Bldg., Dallas.
Kahn, Samuel H., Med. Arts Bldg., Dallas.
Kakaska, George H., 4319 Oak Lawn, Dallas.
Kamholz, Jack H., 712 N. Washington, Dallas.
Kantor, Herman I., 712 N. Washington, Dallas.
Kaplan, Norman M., (Mil.),
5823 Greenlawn, Bethesda, Md.
Katz, Sol M., 800 N. Washington, Dallas.
Kee, John Lester, Jr., 3810 Swiss Ave., Dallas.
Keene, Albert H., 3707 Gaston, Dallas.
Keglovits, Michael J.,
2020 W. Grauwyler, Irving.
Keilson, Gerald R., (Int.),
Methodist Hosp., Dallas.
Kelly, Thomas E., 3520 Greenville, Dallas.
Kent, James M., 9323 Garland Rd., Dallas.
Kerr, Jack G., 1610 Med. Arts Bldg., Dallas.
Keyser, Lester L., SMU Health Cen., Dallas.
Kidd, Frank H., Jr., 4000 Junius St., Dallas.
Kilgore, Bassett B., 3500 Gaston Ave., Dallas.
Kilgore, Donald G., Sr.,
3988 N. Express Way, Dallas.
Kimmerling, Harold W.,
3225 Turtle Creek, Dallas.
Kimsey, Larry R.,
330 N. W. 2nd, Grand Prairie.
Kindley, George C., 3116 Live Oak, Dallas.
King, Carey G., Jr., 3707 Gaston Ave., Dallas.
King, Karl B., 3707 Gaston, Dallas.
King, Virgil M., 1301 Elm St., Carrollton.
Kipp, Dean C., 3601 Swiss, Dallas.
Kirksey, Thomas M., Med. Arts Bldg., Dallas.
Klein, Donald L.,
25 Richardson Hgts., Richardson.
Kleinsasser, Leroy J., 3707 Gaston, Dallas.
Knapp, Joseph L.,
210 N. Westmoreland, Dallas.
Knickerbocker, Bruce A., 3403 Hall St., Dallas.
Knight, Marvin P., 2618 Welborn, Dallas.
Knowles, W. Mood, 8215 Westchester, Dallas.
Koch, William T., 6331 Prospect, Dallas.
Korkmas, Maurice V.,
2018 W. Grauwyler, Irving.
Kozlowski, Eufrozyna J.,
4500 S. Lancaster, Dallas.
Kowierschke, Sidney W., (Int.),
St. Pauls Hosp., Dallas.
Krakusin, Jacob S.,
109 Walnut Hill Village, Dallas.
Kraus, William L., 3710 Swiss Ave., Dallas.
Krebs, David E.,
525 W. Pleasant Run, Lancaster.
Kregel, Louis A., 3707 Gaston Ave., Dallas.
Krempin, Herman O., 1511 N. Beckley, Dallas.
Kresh, Herbert, 9105 Diceman Drive, Dallas.
Kreymer, George C.,
1322 Med. Arts Bldg., Dallas.
Krusen, Edward M., Jr., Baylor Hosp., Dallas.
Krusen, Ursula M. L.,
3500 Coalgate Ave., Dallas.
Kugler, Joseph S., 109 S. Delaware, Irving.
Kurilecz, Michael, 8215 Westchester, Dallas.
Kurilecz, Peter, Jr.,
635 Med. Arts Bldg., Dallas.
Lair, Nard, 10710 Shiloh Rd., Dallas.
Laird, Paul C., Box 515, Irving.
Lancaster, Mary Agnes,
1434 Med. Arts Bldg., Dallas.
Landress, J. Byron, 203 S. 8th, Garland.
Langley, Welborn J.,
10809 Garland Rd., Dallas.
Langston, William G., 3707 Gaston, Dallas.
Lanier, Jack E., 9528 Lemmon Ave., Dallas.
Lanius, John W., 220 Med. Arts Bldg., Dallas.
Lankford, Livius L., 4105 Live Oak, Dallas.
La Prade, William H.,
9323 Garland Rd., Dallas.
LaRue, Kenneth W., 1001 St. Joseph, Dallas.
Lauderdale, Robert A., (Int.),
Methodist Hosp., Dallas.
Laugenour, Dudley P., 4338 Lemmon, Dallas.
Launey, George V., 4536 S. Lindhurst, Dallas.
Lee, Dorothy, (Int.),
St. Pauls Hospital, Dallas.
Lee, Ridings E., 3606 Maple Ave., Dallas.
Legg, Eugene P., 3707 Gaston, Dallas.
Leggett, L. Waldo, 233 W. 10th, Dallas.
Lehman, Irwin, (Int.),
Parkland Mem. Hosp., Dallas.
Leib, Luis, (Mil.),
ARDC Holliman AFB, Alamogordo, N.M.
Leland, Glen V., Jr.,
110-G Walnut Hill Village, Dallas.
LeMaistre, Charles A., 3819 Maple Ave., Dallas.
LeMay, Sonley R., Jr., (Mil.),
USA Disp., Ft. Sam Houston.
Lemmon, Mark L.,
1122 Med. Arts Bldg., Dallas.
Leon, Robert L., 5323 Harry Hines, Dallas.
Letteri, Michael F., (Int.),
St. Paul Hosp., Dallas.
Leventhal, Milton, (Int.),
Parkland Mem. Hosp., Dallas.
Levin, Paul M., Med. Arts Bldg., Dallas.
Lewis, Elbert H.,
Wynnewood Prof. Bldg., Dallas.
Lewis, Jerry M., 1420 Med. Arts Bldg., Dallas.
Lieberman, Philip H., (Int.),
993 E. 8th St., Brooklyn, N. Y.
Lieberman, Zelig H.,
712 N. Washington, Dallas.
Liebes, George J., Jr., 8215 Westchester, Dallas.
Lifson, William L., (Mil.),
Tripler Army Hosp., Honolulu, Hawaii.
Light, Flominda, 7306 Ferguson, Dallas.
Liles, John H., Jr.,
705 W. Randol Mill Rd., Arlington.
Lindsay, Joseph H.,
315 W. Preston For., Dallas.
Lindsey, William H., P. O. Box 2699, Dallas.
Linsey, Ralph M.,
Rockwall Med. Hosp., Rockwall.
Lippas, John, 1717 Pacific, Dallas.
Livingston, Shields O.,
712 N. Washington, Dallas.
LoBello, Leon C., 3601 Swiss Ave., Dallas.
Lodowski, Charles H.,
2317 Mt. Lake Rd., Dallas.
Loeb, Ellen, 4318 Briar Creek, Dallas.
Loftis, Earl L., 3707 Gaston, Dallas.
Loiselle, Albert O., 2601 Welborn, Dallas.
Long, Gerald D., 4338 Lemmon, Dallas.
Long, Troy F., 1510 Med. Arts Bldg., Dallas.
Looney, W. W., 738 W. 10th St., Dallas.
Love, Horace G., Jr., (Mil),
U. S. Naval Hosp., Portsmouth, Va.
Lowry, James S., 3601 Swiss, Dallas.
Lu, Jen Lung, 201 W. 10th, Dallas.
Ludden, Keene F., 111 E. Woodin, Dallas.
Luecke, P. E., Sr., Box 28, Dallas.
Luecke, Percy E., Jr., 4105 Live Oak, Dallas.
Luhn, Nellie R., Parkland Hosp., Dallas.
Lukeman, Herman J.,
9323 Garland Rd., Dallas.
Lumpkin, Forrest E., Jr.,
8215 Westchester, Dallas.
Lumpkin, Walter L., (Mil), Dallas.
Lyday, Victor I., 516 Med. Arts Bldg., Dallas.
MacDonald, Paul C., (Int.),
Parkland Mem. Hosp., Dallas.
Machen, Robert N.,
Wynnewood Prof. Bldg., Dallas.
Madison, Leonard L., 2211 Oak Lawn, Dallas.
Maffett, Minnie L.,
416 Med. Arts Bldg., Dallas.
Magee, Robert V. G.,
2606 S. Garland Rd., Garland.
Magers, Morris E., 4105 Live Oak, Dallas.
Mahaney, Charles L., 6003 Victor, Dallas.
Mahon, Ralph D., Jr., 6003 Victor, Dallas.
Mallams, John T., 712 N. Washington, Dallas.
Maniotis, James G., 811 S. Windomere, Dallas.
Marchman, Oscar M., Jr.,
1314 Med. Arts Bldg., Dallas.
MarDock, Julian, 3418 S. Beckley, Dallas.
Marshall, James H., (Hon.),
6006 Stefani, Dallas.
Martin, Charles L., 3501 Gaston Ave., Dallas.
Martin, Jack, 5323 Harry Hines, Dallas.
Martin, James A., 3501 Gaston, Dallas.
Martin, John G., 1511 N. Beckley, Dallas.
Martin, Thomas A., Jr.,
832 Med. Arts Bldg., Dallas.
Martin, Wayne E., 10023 Garland Rd., Dallas.
Martinak, Richard E., 4206 Swiss, Dallas.
Martinez, Daniel M.,
174 Wynnewood Prof., Dallas.
Mason, Eugene E., 3707 Gaston Ave., Dallas.
Mason, Porter K., Med. Arts Bldg., Dallas.
Massad, Eugene M.,
13545 Webbs Chapel Rd., Dallas.
Mathews, Paul W., (Hon.),
5951 Sherry Lane, Dallas.
Mathis, John P., 712 N. Washington, Dallas.
Mattson, Harold A., Box 4296, Dallas.
Mauk, Ferald D.,
Box 6210, Terminal Annex, Dallas.
Maxfield, G. S. Jack,
2711 Oak Lawn Ave., Dallas.
Maxfield, James R., Jr.,
2711 Oak Lawn Ave., Dallas.
Maxfield, William S., (Int.),
Johns Hopkins Hosp., Baltimore, Md.
Maxwell, Hal W., 624 Med. Arts Bldg., Dallas.
May, James L., 212 S. Main, Irving.
May, James S., Jr., 3113 Routh St., Dallas.
Mayfield, Imogene, 121 S. Zangs, Dallas.
McAlister, James R.,
532 Med. Arts Bldg., Dallas.
McAnally, Billie H.,
2341 Gus Thomasson, Dallas.
McBee, James W., (Int.), V. A. Hosp., Dallas.
McBride, Dayton C., 3330 Douglas St., Dallas.
McBride, Robert B., 3330 Douglas St., Dallas.
McCall, Robert A., 410 Med. Arts Bldg., Dallas.
McCarley, Ben P., 203 W. Tyler, Richardson.
McCarville, John R., Bryan.
McCauley, H. Leake, Jr., (Int.),
1028 W. Boyd, Norman, Okla.
McCleskey, Otris L., (Int.),
Baylor Med. Center, Dallas.
McClung, Hugh L.,
1526 Med. Arts Bldg., Dallas.
McCreary, Howell S., (Int.),
3500 Gaston Ave., Dallas.
McCrory, Thomas M., 3707 Gaston, Dallas.
McCullough, John H.,
182 Casa Linda Pla., Dallas.
McDonald, Viola Y.,
1449 San Rafael Drive, Dallas.
McFarland, Gordon B.,
3701 Fairmount, Dallas.
McGinnis, Albert, (Int.),
109 Walnut Hill Village, Dallas.
McGuire, Donald E., Jr.,
712 N. Washington, Dallas.
McGuire, Joseph H., (dead), Dallas.
McIver, Julius, 4029 Lemmon, Dallas.
McKinney, James M., 8215 Westchester, Dallas.
McLaurin, Hugh L., (Hon.),
5036 Seneca Drive, Dallas.
McNamara, James C., Jr.,
5221 Locksley, Dallas.
McNeill, Arch J., Med. Arts Bldg., Dallas.
McNeill, Joseph P., 3707 Gaston, Dallas.
McRee, Cecil A., (Int.), Baylor Hosp., Dallas.
McReynolds, Benjamin A.,
3707 Gaston Ave., Dallas.
Meisenbach, Albert E., Jr.,
317 Med. Arts Bldg., Dallas.
Mendel, E. B., 3702 Worth St., Dallas.
Mendenhall, Elliott,
1217 Med. Arts Bldg., Dallas.
Merrick, Ben A., 3520 Cedar Springs, Dallas.
Metz, M. Hill, 4319 Oak Lawn, Dallas.
Mewhinney, Logan U., 6115 Lavista, Dallas.
Michael, Ludwig A., 3707 Gaston Ave., Dallas.
Miller, Alan N., II, 2028 W. Grauwyler, Irving.
Miller, Donald B.,
1005 SW 3rd St., Grand Prairie.
Miller, Ed Crow, 8215 Westchester, Dallas.
Miller, Ervin Ray, 3330 S. Lancaster, Dallas.
Miller, J. E., 6407 Forest Lane, Dallas.
Miller, John F., 1511 N. Beckley, Dallas.
Miller, Tate, Med. Arts Bldg., Dallas.
Miller, William F., S. W. Med. Schl, Dallas.
Mills, E. Grady, 2353 Gus Thomasson, Dallas.

Mills, James T., 3707 Gaston, Dallas.
Millwee, Robert H.,
1400 Stemmons Ave., Dallas.
Mims, Harold M., 3707 Gaston Ave., Dallas.
Minnett, John S., 2929 Welborn, Dallas.
Mitchel, Ben F., 3707 Gaston Ave., Dallas.
Mitchell, Harry J., 915 St. Joseph, Dallas.
Mitchell, Joseph D., Jr., 3707 Gaston, Dallas.
Mitz, Robert, 308 NW 2nd, Grand Prairie.
Mobley, Henry B., (Int.),
2345 52nd St., Dallas.
Monahan, Eugene T., 3330 S. Lancaster, Dallas.
Montgomery, Henry G., Box 28, Dallas.
Montgomery, John C., 911 St. Joseph, Dallas.
Montgomery, Philip O'B.
9039 Devonshire, Dallas.
Moody, Joe V., 1511 N. Beckley, Dallas.
Mooney, Ken, 1528 Med. Arts Bldg., Dallas.
Moore, Halcuit, 4105 Live Oak, Dallas.
Moore, Kendall H., 9323 Garland Rd., Dallas.
Moore, Ramsay H., 8215 Westchester, Dallas.
Moore, Robert L., 3403 Hall St., Dallas.
Moore, William T., 3911 Maple Ave., Dallas.
Morgan, Bill C., 6003 Victor, Dallas.
Morris, A. Truett, 4224 Swiss Ave., Dallas.
Morris, Charles R., 8215 Westchester, Dallas.
Morris, Donald P., 911 St. Joseph, Dallas.
Morrison, Donald D., 6206 Lavendale, Dallas.
Mueller, Helmut A.,
813 N. Zangs Blvd., Dallas.
Mullikin, Gerald G., 4206 Swiss Ave., Dallas.
Munsell, Donald W., 3607 Gaston, Dallas.
Murillo, Sergio L., 3116 Fairmount, Dallas.
Murley, Warren T., Jr., (Mil.),
Box 621, Harlingen AFB.
Murphy, Joseph B., 4101 Hanover, Dallas.
Murphy, Thomas P., Jr., (Int.),
Childrens Med. Center, Dallas.
Murray, Hugh D., 317 N. Zangs, Dallas.
Mustain, Rhoads, 3607 Gaston Ave., Dallas.
Nabors, G. Cooley, 9429 El Centro, Dallas.
Nash, Gloria H., 1100 N. Canterbury, Dallas.
Nash, Tom M., 729 Med. Arts Bldg., Dallas.
Neal, Leroy J., (Mil.),
U. S. Naval Depot, Seal Beach, Calif.
Needham, Perry Q., Med. Arts Bldg., Dallas.
Neel, Joseph C., Med. Arts Bldg., Dallas.
Nelson, Arthur G., 9323 Garland Rd., Dallas.
Nelson, Marion H., (Int.),
1611 Ridgemont, Austin.
Nelson, Oscar T., 6003 Victor, Dallas.
Nesbit, Harold T., 3707 Gaston Ave., Dallas.
Nesbitt, Irene T., 4335 Lemmon, Dallas.
Neuman, Albert, 3829 Hall, Dallas.
Newell, Edward A., 1511 N. Beckley, Dallas.
Newell, Philip D., 930 N. Edgefield, Dallas.
Newsom, Asa A., 3707 Gaston, Dallas.
Newsom, Asa A., Jr., 3707 Gaston Ave., Dallas.
Newton, Frank H., 209 Med. Arts Bldg., Dallas.
Niblo, Grady, Jr., 351 W. Jefferson, Dallas.
Nicolaou, George T., 911 St. Joseph, Dallas.
Nitsche, Ernest W., (Hon.),
5749 Gaston, Dallas.
Noonan, Richard L.,
111 N. E. 11th St., Grand Prairie.
Norman, Floyd A., 4143 Cole Ave., Dallas.
Noteboom, Gerard,
1142 Edgefield Ave., Dallas.
Nunnelley, Emmett C., Jr.,
3219 Turtle Creek, Dallas.
Oberlin, Donald L., (Int.),
Akron City Hosp., Akron, Ohio.
O'Brien, Harold A., 3707 Gaston, Dallas.
O'Brien, Justin D.,
1135 Med. Arts Bldg., Dallas.
Ogle, Joseph C., 737 N. Bishop, Dallas.
Olinger, Sheff D., Jr.,
712 N. Washington, Dallas.
Oliver, Billy Byrd, 3707 Gaston Ave., Dallas.
Ossenfort, William F.,
5851 Del Roy Drive, Dallas.
Ossenfort, Wm. F., Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Overton, Philip M., (Int.),
Parkland Hosp., Dallas.
Pace, John M., 428 Med., Arts Bldg., Dallas.
Palmer, Robert B., 3707 Gaston Ave., Dallas.
Park, Barton E., 1121 W. Jefferson, Dallas.
Parker, Edward R., 6115 La Vista, Dallas.
Parnell, Billy Joe,
1221 Med. Arts Bldg., Dallas.
Passamonte, Jane A.,
1516 W. Jefferson, Dallas.
Paternostro, Chas. J.,
Med. Arts Bldg., Dallas.
Patman, R. Donald, (Int.),
Parkland Mem. Hosp., Dallas.
Patterson, Casey E.,
729 Med. Arts Bldg., Dallas.
Patterson, Cecil O.,
1414 Med. Arts Bldg., Dallas.
Patton, Walter H., 6003 Victor, Dallas.
Paulson, Donald L., 3810 Swiss Ave., Dallas.
Pauly, A. Nottingham,
1035 N. Zangs Blvd., Dallas.
Payne, Frank C., Jr., (Int.),
6046 Sherry Lane, Dallas.
Payne, Virgil M., Jr.,
1209 Med. Arts Bldg., Dallas.
Pearcy, Frank, 3929 Turtle Creek, Dallas.
Peden, James K., 1420 Med. Arts Bldg., Dallas.
Pence, Ludlow M.,
210 N. Westmoreland, Dallas.
Perkins, Jack F. (Hon.),
3526 Cedar Springs, Dallas.
Perrin, Robert W., 8215 Westchester, Dallas.
Perryman, Ray W.,
746 S. Central Expy., Richardson.
Peyton, John B., Med. Arts Bldg., Dallas.
Pharo, Milam B., 8215 Westchester, Dallas.
Phillips, James R.,
308 N. W. 2nd St., Grand Prairie.
Phillips, Roycerene H.,
308 N. W. 2nd St., Grand Prairie.
Phillips, Sam H., Jr., 3707 Gaston Ave., Dallas.
Pickard, James M., 1936 Amelia, Dallas.
Pickett, Taylor T., 5702 Goliad, Dallas.
Pickett, William H.,
922 Med. Arts Bldg., Dallas.
Pickle, Coy R.,
1316 W. Garland Ave., Garland.
Pierce, Graham L., 4208 Swiss Ave., Dallas.
Pierce, Robert J., 109 S. Delaware, Irving.
Pierson, Milton A., 9323 Garland Rd., Dallas.
Pinkston, Lee G., 3305 Thomas Ave., Dallas.
Piranio, Joe C., 606 Monssen Drive, Dallas.
Pirrung, Joey M., Box 135, Mesquite.
Pizette, Murray, (Int.),
9323 Garland Rd., Dallas.
Popkess, Fred G., 302 Preston Royal, Dallas.
Porter, George L., 3707 Gaston, Dallas.
Porter, Louis H., II,
712 N. Washington, Dallas.
Portman, Robert K., 737 N. Bishop, Dallas.
Potts, James M., (Hon.), 4100 Caruth, Dallas.
Potts, Robert J., P. O. Box 900, Dallas.
Potts, William H., Jr., Box 28, Dallas.
Poulos, Ernest, 4429 Windsor, Dallas.
Powell, Dudley V., 4207 Oakland, Dallas.
Powell, Homer, (Hon.),
2635 W. Mulberry, San Antonio.
Powell, Jay W., 2024 W. Grauwyler, Irving.
Powell, William J.,
McKinney At Main, Richardson.
Powers, Hugh W. S.,
8215 Westchester, Dallas.
Pratt, Edward L., 3721 N. Harwood, Dallas.
Prejean, Oran V.,
1317 N. Washington, Dallas.
Preuss, Fred S., 3607 Gaston Ave., Dallas.
Price, Harry S., 351 W. Jefferson, Dallas.
Pritchard, Jack A., 5323 Harry Hines, Dallas.
Puls, Richard J., 610 Med. Arts Bldg., Dallas.
Quinn, Lester H., 4020 Junius, Dallas.
Rabinowitz, Haskell I., (Int.),
3534 Maple, Dallas.
Race, George J., 712 N. Washington, Dallas.
Railsback, George D., Jr.,
2011 W. Irving Blvd., Irving.
Ramsey, John D., (Int.),
Parkland Hosp., Dallas.
Randel, Joseph C., (Int.),
Parkland Mem. Hosp., Dallas.
Range, N. Haskell, 712 N. Washington, Dallas.
Rao, Mysore N., 4227 Herschel, Dallas.
Rattan, Paul M., Med. Arts Bldg., Dallas.
Ray, James H., (Hon.), Lewisville.
Read, Edwin P.,
117 N. E. 8th St., Grand Prairie.
Reagan, A. Morriss,
110H Walnut Hill Village, Dallas.
Regnier, Thomas A., 135 W. 10th St., Dallas.
Reid, Allen F., 3145 Spur Trail, Dallas.
Reisman, David D., 3707 Gaston Ave., Dallas.
Renken, Harry J., Jr., 3707 Gaston, Dallas.
Reuss, G. Thomas, 901 Texas Bk. Bldg., Dallas.
Reynolds, Claude E., (Mil.),
21503 Ct. APO 50, San Francisco, Calif.
Reynolds, Jack, Parkland Mem. Hosp., Dallas.
Reynolds, Rolland C., (Int.),
Parkland Mem. Hosp., Dallas.
Reynolds, William S.,
1537 Med. Arts Bldg., Dallas.
Rhodes, Edward L.,
1728 Charlton, Ann Arbor, Mich.
Richardson, Edward R.,
220 Med. Arts Bldg., Dallas.
Richburg, Paul L., 3707 Gaston Ave., Dallas.
Richmond, Marion B., 2525 Centerville, Dallas.
Ricketts, Marion M.,
Wynnewood Prof. Bldg., Dallas.
Riddle, Penn, (Hon.),
964 Sam Dealy Drive, Dallas.
Riedel, Albert F., Jr., (Int.),
P. O. Box 1769, Dallas.
Rippy, Edwin L., 3622 Fairmount, Dallas.
Ritchey, Lloyd F., 8215 Westchester, Dallas.
Roach, Joe G., Jr., 6046 Sherry Lane, Dallas.
Robbins, Jacob H., 354 Hillside Village, Dallas.
Roberts, Albert D., Jr., (Int.),
510 Med. Arts Bldg., Dallas.
Roberts, Joe H., 101 Iowa St., Irving.
Roberts, Tom Ray, 6331 Prospect, Dallas.
Robins, Keith I., 2710 Valley View, Dallas.
Robinson, David L., (Mil.),
855th Med. Group, Barksdale AFB, La.
Robinson, Fabian J., (Inac.),
6411 Bandera, Dallas.
Robinson, Wayne T., 3814 Fairmount, Dallas.
Robison, Leonard J., 6003 Victor, Dallas.
Rogers, Fred T., Box 28, Dallas.
Rogers, Gene W., 3330 S. Lancaster, Dallas.
Rogers, Harriet N., 1936 Amelia St., Dallas.
Rogers, Paul A., 806 Med., Arts Bldg., Dallas.
Romero, Robert, (Int.),
Parkland Mem. Hosp., Dallas.
Rosenthal, Raoul S., (dead), Dallas.
Ross, Edward S., 2929 Welborn, Dallas.
Ross, James K., 208 Med. Arts Bldg., Dallas.
Rosser, Curtice, 710 Med. Arts Bldg., Dallas.
Rothschild, J. E., 3707 Gaston Ave., Dallas.
Rounsaville, John Q. 504 W. 10th, Dallas.
Rountree, John R.,
4 Richardson Hts., Richardson.
Rouse, Milford O.,
1414 Med. Arts Bldg., Dallas.
Rowe, J. Forsythe, (Hon.),
1402 Kings Highway, Dallas.
Rowe, Robert J., 3707 Gaston Ave., Dallas.
Royer, Emmett M., 9323 Garland Rd., Dallas.
Russell, Melvin G., 427 W. 10th, Dallas.
Russell, Richard R., 4006 Urban, Dallas.
Sacher, Clarence B.,
817 Med. Arts Bldg., Dallas.
Saldivar, Julian T., 3300 Cole Ave., Dallas.
Salem, Samuel D.,
Wynnewood Prof. Bldg., Dallas.
Sams, Lewis C., 201 W. 10th St., Dallas.
Sanders, Morris, 3607 Gaston Ave, Dallas.
Sanders, Wilford M., Jr.,
721 Med. Arts Bldg, Dallas.
Sazama, John J., 3707 Gaston, Dallas.
Scales, John G., 400 W. 9th St., Dallas.
Scanland, Viola P., 623 Med. Arts Bldg., Dallas.
Schaefers, J. G., Jr.,
8215 Westchester, Dallas.
Schenewerk, George A.,
8215 Westchester, Dallas.
Schneider, Peter W., 427 W. 10th St., Dallas.
Schnitzer, Ben, (Int.),
Parkland Mem. Hosp., Dallas.
Schnitzer, Bernard, 10622 Garland Rd., Dallas.
Schoch, Arthur G., Med. Arts Bldg., Dallas.
Schonfeld, Murry D., 1203 Gordon St., Dallas.
Schorlemer, Wendell C., (Mil.),
USAF Hosp., Keesler AFB, Miss.
Schreiber, Gus, Jr., 4217 Herschel, Dallas.
Schroeder, Charles F.,
1810 S. Westmoreland, Dallas.
Schroeder, Glenn E., (Int.),
Methodist Hosp., Dallas.
Schuett, Albert J., (Hon.), (Dead), Dallas.
Schwenkenberg, Arthur J.,
210 N. Westmoreland, Dallas.
Scurry, Maurice M., 3710 Swiss, Dallas.
Sears, Alvin D., 3552 Villanova, Dallas.
Seay, Frank O., 220 Med. Arts Bldg., Dallas.
Sebastian, F. J., 3707 Gaston Ave., Dallas.
Seely, M. Stuart, 3911 Gaston Ave., Dallas.
Seidel, Clifford C.,
Wynnewood Med. Bldg., Dallas.
Seldin, Donald W., 2211 Oak Lawn, Dallas.
Selecman, Frank A.,
410 Med. Arts Bldg., Dallas.
Sellars, William A., 638 W. 10th St., Dallas.
Sellers, Lyle M., 3707 Gaston, Dallas.
Sellman, Willard C.,
922 Med. Arts Bldg., Dallas.
Semones, Earl L., (Int.),
1360 N. Lakeshore Dr., Chicago, Ill.
Sewell, James W.,
221 Pleasant Gr. S. C., Dallas.
Shane, J. Howard,
1421 Med. Arts Bldg., Dallas.
Shannon, Arthur W., Jr.,
220 Med. Arts Bldg., Dallas.
Shannon, Hall, 808 Southland Cen., Dallas.
Shannon, Manning B., 4000 Junius, Dallas.
Sharp, Doyle L., 3330 S. Lancaster, Dallas.
Shaw, Marie L., 712 N. Washington, Dallas.
Shaw, Robert R., 3810 Swiss, Dallas.
Shelburne, Samuel A., 3707 Gaston, Dallas.
Sheldon, Lawrence B., Med. Arts Bldg., Dallas.
Shelmire, J. Bedford,
1410 Med. Arts Bldg., Dallas.
Shelmire, J. Bedford, Jr.,
1410 Med. Arts Bldg., Dallas.
Shelton, George L., Jr., 4303 Oakland, Dallas.
Shelton, William P., 8215 Westchester, Dallas.
Shepard, Marvin G.,
722 Med. Arts Bldg., Dallas.

Shinn, Bonner L., 1326 Stemmons Ave., Dallas.
Shires, George Thomas,
5323 Harry Hines, Dallas.
Shires, Robbie Jo, 10023 Garland Rd., Dallas.
Shoecraft, Warren A., 135 W. 10th St., Dallas.
Short, Robert F., Med. Arts Bldg., Dallas.
Shortal, William W., 4217 Swiss, Dallas.
Shropulos, George P., (Int.),
Parkland Mem. Hosp., Dallas.
Shuster, Donald W., 4105 Live Oak, Dallas.
Sibley, George W., 1217 N. Clinton, Dallas.
Siebel, Eldon K., 201 W. 10th St., Dallas.
Siegel, Robert M.,
110-D Walnut Hill Village, Dallas.
Sigel, Zundel, 1900 Forest Ave., Dallas.
Sigler, Howard Y., 4217 Swiss, Dallas.
Sikora, David, 2710 Valley View, Dallas.
Sills, E. Garrett, V. A. Hosp., Dallas.
Simmons, Belvin A., (Int.),
P. O. Box 1769, Dallas.
Simmons, Charles E.,
Southwest Med. Schl., Dallas.
Simmons, Robin E., Box 248, Richardson.
Singleton, John D., 3704 Dickason, Dallas.
Siperstein, Marvin D.,
5323 Harry Hines, Dallas.
Slaughter, B. Celia, Box 28, Dallas.
Slaughter, Manuel O.,
2011 W. Irving Blvd., Irving.
Sloan, Charles M., 206 W. Colorado, Dallas.
Smale, John S., 2722 Shelby Court, Garland.
Small, Andrew B., 3707 Gaston, Dallas.
Smart, Don M., 9323 Garland Road, Dallas.
Smith, Alice L., 3326 Blackburn, Dallas.
Smith, C. Robt., 9323 Garland Rd., Dallas.
Smith, Charles L.,
2806 Fairfax Dr., Garland.
Smith, Elsie A., 4922 Greenville, Dallas.
Smith, Lois W., 3524 Fairmount, Dallas.
Smith, Louis S., P. O. Box 57, Dallas.
Smith, Ralph C., V. A. Hosp., Dallas.
Smith, Richard M., Box 28, Dallas.
Smith, Robert W.,
7805 Lake June Rd., Dallas.
Smith, Sydnie G., 3707 Gaston Ave., Dallas.
Smith, Tom E., 2600 Welborn, Dallas.
Smith, Vinny L., (Hon.),
3337 Mockingbird, Dallas.
Smith, Wright K.,
221 Pleasant Grove S. C., Dallas.
Somer, Joseph, (Int.),
3707 Gaston Ave., Dallas.
Sorrells, Chas. C., (Hon.),
619 N. Mont Clair, Dallas.
Sowers, Harry B., 4618 San Jacinto, Dallas.
Sparkman, Robert S.,
1004 N. Washington, Dallas.
Sparr, Richard A.,
10801 Garland Rd., Dallas.
Speegle, Jackson H.,
210 N. Westmoreland, Dallas.
Speegle, Robert E.,
1002 N. Marion Dr., Garland.
Spegal, Doris V., 6115 La Vista, Dallas.
Spence, Harry M., 4105 Live Oak, Dallas.
Stack, John W., Box 135, Mesquite.
Standlee, Earle, 3535 W. Lawther Dr., Dallas.
Stanley, E. Stephen,
610 Preston Forest Cen., Dallas.
Stanton, Elbert H., 3400 Irving Blvd., Irving.
Stapp, William F., (Int.),
1912 E. Beach St., Gulfport, Miss.
Stayer, David S.,
606 Med. Arts Bldg., Dallas.
Stayer, Glenn C., 427 W. 10th St., Dallas.
Stayer, Irene M., Box 295, Irving.
Steil, Cecil I., 3617 Fairmount, Dallas.
Stembridge, Vernie A.,
5323 Harry Hines, Dallas.
Steph, Donal W., (Int.),
214 E. Houston, Tyler.
Stephenson, James H., (Hon.),
6932 Lavendale, Dallas.
Stephenson, Martin L., Jr.,
4500 S. Lancaster, Dallas.
Stephenson, W. O., (Hon.),
4005 Hall, Dallas.
Stevenson, Stanley L., 5221 Locksley, Dallas.
Stewart, Robert D., 135 Melba, Dallas.
Stiles, Wendel A., Med. Arts Bldg., Dallas.
Strauss, Elias, 3707 Gaston, Dallas.
Streitmatter, David E.,
302 Preston Royal, Dallas.
Strickland, Wm. M., Jr.,
Parkland Mem. Hosp., Dallas.
Stringer, Charles F.,
746 S. Central Expressway, Richardson.
Strong, James C.,
1213 Walnut St., Wilmington, Dela.
Strother, Wm. K., Jr.,
1311 N. Washington, Dallas.
Stuart, John A., Jr.,
712 N. Washington, Dallas.
Stubblefield, Robt. L.,
5323 Harry Hines, Dallas.
Super, Archie R.,
418 Med. Arts Bldg., Dallas.
Susong, William A., (Int.),
Parkland Mem. Hosp., Dallas.
Sutherland, Dan R., 2618 Welborn, Dallas.
Sutherland, Donald A.,
4033 Lawther Dr., Dallas.
Suttle, R. Courtney,
3406 McFarlin Blvd., Dallas.
Sweeney, J. Shirley,
4005 St. Andrews Dr., Dallas.
Talkington, Perry C.,
1420 Med. Arts Bldg., Dallas.
Tandy, Charles C., 206 W. Colorado, Dallas.
Taylor, H. Earl,
220 Med. Arts Bldg., Dallas.
Taylor, Harold F.,
S.W. Med. School, Dallas.
Taylor, Vann S., 712 N. Washington, Dallas.
Teel, Theodore T., Jr.,
221 Pleasant Grove S. C., Dallas.
Temple, Bobby L., (Int.),
3330 Douglas St., Dallas.
Teng, Hsi Ching, 3707 Gaston Ave., Dallas.
Terry, J. Glenn, 1001 St. Joseph, Dallas.
Terry, Jack S.,
220 Med. Arts Bldg., Dallas.
Thomas, Harold R., 9323 Garland Rd., Dallas.
Thomas, Paul J., 3707 Gaston, Dallas.
Thomas, W. Maxwell, 3707 Gaston, Dallas.
Thomasson, A. R., Jr., Med. Arts Bldg., Dallas.
Thomasson, A. R., Sr., (Hon.),
Med. Arts Bldg., Dallas.
Thompson, Barrett M., Box 686, Irving.
Thompson, Jesse E.,
1008 N. Washington, Dallas.
Thompson, L. S., 726 Med. Arts Bldg., Dallas.
Thompson, L. S., Jr.,
726 Med. Arts Bldg., Dallas.
Tigertt, W. D., (Mil.), Dallas.
Timken, Kenneth R., (Int.),
St. Paul Hospital, Dallas.
Tirmenstein, Martine G.,
206 W. Colorado, Dallas.
Tittle, Guy A., 3600 Fairmount, Dallas.
Tobey, Nathan G., 521 N. Washington, Dallas.
Tobolowsky, Dave,
Wynnewood Prof. Bldg., Dallas.
Tocker, Albert M., 8215 Westchester, Dallas.
Tomkies, James S., Med. Arts Bldg., Dallas.
Tomlinson, Jack R., 233 W. 10th, Dallas.
Tompsett, Ralph R., 3500 Gaston Ave., Dallas.
Touchstone, Jay L.,
606 Med. Arts Bldg., Dallas.
Tromly, Robert G., 135 Melba, Dallas.
Tschumy, William O., Jr.,
1616 Med. Arts Bldg., Dallas.
Tsukahara, William, 1400 Forest Ave., Dallas.
Tubb, Cullen L., 220 Med. Arts Bldg., Dallas.
Turbeville, Louis R., 9323 Garland Rd., Dallas.
Uhler, Claude, 226 S. Edgefield, Dallas.
Ulevitch, Herman, 915 St. Joseph, Dallas.
Underwood, George M., Box 28, Dallas.
Vanarta, John C., III,
Southwestern Medical School, Dallas.
Van Burkleo, Julia B., (Int.),
4804 Manette St., Dallas.
Van Cleave, Charles E., 202 W. 10th, Dallas.
Vanderpool, B. David, Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Vardy, Richard L., (Int.),
Parkland Mem. Hosp., Dallas.
Vassallo, Alfred L., 6738 Glendora, Dallas.
Vassallo, Harry R., 4101 Lemmon, Dallas.
Veninga, Frederick W.,
3520 Cedar Springs, Dallas.
Vermooten, Vincent, 3607 Gaston Ave., Dallas.
Vieaux, Jules W., 712 N. Washington, Dallas.
Vineyard, John P., Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Votteler, Theodore P., 3524 Fairmount, Dallas.
Vowell, Billy S., (Int.),
109 S. Delaware, Irving.
Waddell, O. Jay, 220 Med. Arts Bldg., Dallas.
Wade, T. W., 4500 Lancaster, Dallas.
Wagner, Wilson O., Carrollton.
Wagoner, Roy D., 182 Casa Linda Pl., Dallas.
Waldman, Morris F.,
5414 W. University, Dallas.
Waldron, W. Doyle, 1315 W. Jefferson, Dallas.
Walker, Jack Earl, (Inac.),
Baptist T.B. Hosp., Mbeya, East Africa.
Walker, Price M., (Hon.),
2225 Bellefontaine, Houston.
Walker, Tommy R., 3607 Gaston Ave., Dallas.
Wallace, Gordon K., Med. Arts Bldg., Dallas.
Warden, Don P., (Int.),
5007 Wenonah Dr., Dallas.
Ware, Elgin W., Jr., 3707 Gaston, Dallas.
Ware, F. Leon, 2618 Welborn, Dallas.
Warkentin, Harold J., 3605 Routh St., Dallas.
Warren, Charles H.,
506 Med. Arts Bldg., Dallas.
Wasserman, Eugene, 3607 Gaston, Dallas.
Waterman, William E., (Int.),
Mass. Mem. Hosp., Boston, Mass.
Watkins, A. B., 725 N. Kaufman, Seagoville.
Watkins, Margaret, 3503 Fairmount, Dallas.
Weary, Willard B., 3607 Gaston, Dallas.
Weatherby, Marvine,
914 Med. Arts Bldg., Dallas.
Webb, Robert W., 4338 Lemmon, Dallas.
Weed, Olga, 2211 Stutz Dr., Dallas.
Weiner, Bernard S.,
113 N. Center, Grand Prairie.
Weiner, David O., 3707 Gaston Ave., Dallas.
Weisz, Stephen, 1637 Med. Arts Bldg., Dallas.
Welch, Mark L., 908 Med. Arts Bldg., Dallas.
Wells, James T., (Hon.),
5350 Vickery Blvd., Dallas.
West, Ann, 808 Med. Arts Bldg., Dallas.
Whalley, Peggy Joyce, (Int.),
Parkland Mem. Hosp., Dallas.
Wharton, Turner A., 1936 Amelia, Dallas.
White, Claud V.,
3535 Cedar Springs Rd., Dallas.
White, Edward, 523 Med. Arts Bldg., Dallas.
White, Edward, Jr., (Int.),
3086 Newcastle, Dallas.
White, Hugh D., (Hon.),
3429 University Blvd., Dallas.
White, Robert F., Med. Arts Bldg., Dallas.
White, Roland L., 10023 Garland Rd., Dallas.
Whitney, David G.,
4922 Greenville Ave., Dallas.
Whitten, Merritt B.,
1430 Med. Arts Bldg., Dallas.
Whittlesey, P. E., Jr.,
Wynnewood Med. Bldg., Dallas.
Wiedeman, John E., SMU Health Cen., Dallas.
Wilder, Felix F., 925 Med. Arts Bldg., Dallas.
Wilke, Joseph E., Jr., 206 W. 10th St., Dallas.
Wilkinson, Wallace B., 101 N. Zangs, Dallas.
Willbanks, Otto L., Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Willeford, Carl A., 111 E. Woodin, Dallas.
Williams, Bryan, 3710 Swiss, Dallas.
Williams, G. Raworth,
616 Med. Arts Bldg., Dallas.
Williams, J. O., Jr., (Mil.),
Dyess AFB, Abilene.
Williams, Joseph R.,
2918 Thomas Ave., Dallas.
Williams, Oscar B., Jr.,
3607 Gaston Ave., Dallas.
Williams, Owen Dale, (Mil.),
3650th USAF Hosp., Webb AFB.
Williams, Paul C., 3607 Gaston, Dallas.
Willis, Kathryn W., 5323 Harry Hines, Dallas.
Wilson, Ben J.,
2232 N. 7th St., Grand Junction, Colo.
Wilson, Bruce F., (Int.),
443 Elmhurst, San Antonio.
Wilson, Charles M.,
712 N. Washington, Dallas.
Wilson, Hugh E., III,
5323 Harry Hines, Dallas.
Winans, Henry M., 2703 Oak Lawn, Dallas.
Winans, Henry M., Jr.,
2703 Oak Lawn Ave., Dallas.
Winborn, Claude D., 3707 Gaston, Dallas.
Windmiller, Joan, (Int.),
Children's Med. Cen., Dallas.
Winkelbauer, Rudolf G., (Mil.),
5th Gen. Hosp., APO 154, N.Y., N. Y.
Winn, Robert E., 8215 Westchester, Dallas.
Winn, Watt W., 2606 Welborn, Dallas.
Wolfe, Joseph, 3609 Cedar Springs, Dallas.
Wolff, Paul M., (Inac.),
6414 Blanch Cir., Dallas.
Wolford, Robert B., (Hon.),
3819 Moler St., Dallas.
Wolfram, Julius, 3707 Gaston Ave., Dallas.
Womack, Jack I., 10753 Preston Rd., Dallas.
Wood, Joe B., 712 N. Washington, Dallas.
Wood, Thomas P., Jr., (Mil.),
Box P, Jacksboro.
Wood, William A.,
9528 Webbs Chapel Rd., Dallas.
Woodard, Gay T., 233 W. 10th St., Dallas.
Woodard, T. Leroy,
207 Med. Arts Bldg., Dallas.
Woodard, William C.,
324 Med. Arts Bldg., Dallas.
Woods, Channing, 4105 Live Oak, Dallas.
Woods, Ozro T., 4105 Live Oak, Dallas.
Woolf, Jack I., 3607 Gaston, Dallas.
Worsham, A. Gordon, 4143 Cole Ave., Dallas.
Wynne, Buck Jim, Jr.,
628 Med. Arts Bldg., Dallas.
Yeager, Henry, Jr., (Int.),
Parkland Mem. Hosp., Dallas.
Yeary, Robert A., 1002 Marion Dr., Garland.
Young, John G., 4005 St. Andrews, Dallas.
Youngblood, J. Wade,
8215 Westchester, Dallas.
Ziff, Morris, 5323 Harry Hines, Dallas.

DENTON

Adami, Gilbert E., 500 W. Oak, Denton.
Boyd, Dickson K., 525 S. Locust, Denton.
Burgess, H. M.,
Normal & Scripture, Denton.
Davis, Bert E., Denton.
Finlay, Alexander M., Jr.,
909 Edgewood St., Denton.
Glass, Paul F., Jr., Normal & Scripture, Denton.
Gochnour, Runnald W., Justin.
Haggard, Scott, 121 Piner St., Denton.
Hayes, Lindley O., Denton.
Holland, Joseph W., 525 S. Locust, Denton.
Jones, James H., Normal & Scripture, Denton.
Kinard, Conrad L.,
Normal & Scripture, Denton.
Lee, Robert Joe, Normal & Scripture, Denton.
Lockwood, Robert M., 526 N. Locust, Denton.
Maddox, Wm. Gordon,
Med. Arts Bldg., Denton.
McClendon, Harry M., Denton.
Miller, Walter S., Jr., 420 Normal, Denton.
Moreland, Virginia L., 526 N. Locust, Denton.
Norgaard, Hal V., 420 Normal, Denton.
Patterson, Thomas V., 502 W. Oak, Denton.
Pedigo, Smith J., 2025 Crestwood, Denton.
Rainone, Frank A., Jr.,
210 N. Poydras, Lewisville.
Remley, William A., 525 S. Locust, Denton.
Schedler, Paul W., 120 W. Oak, Denton.
Schlegel, Harold F.,
220 N. Poydras St., Lewisville.
Shirley, Geraldine W. S., T.S.C.W., Denton.
Short, Bobby J., (Inac.), Denton.
Silverthorne, M. Clark, 526 N. Locust, Denton.
Tatum, James E., Jr., 1403 N. Elm, Denton.
Thomas, J. David, 2017 Brown Dr., Denton.
Weathers, Paul E.,
1005 Decatur Dr., W., Denton.
Wyss, Albert E., 123 Piner St., Denton.

ELLIS

Baker, Edmund F., Ennis.
Calloway, Arthur E.,
712 E. Jefferson, Waxahachie.
Clark, J. Lawrence, 710 N. Preston St., Ennis.
Compton, John G., Jr.,
201 Ferris Ave., Waxahachie.
Curby, John H., (Hon.), Maypearl.
Donnell, Herbert, 507 W. Main, Waxahachie.
Dykes, Arthur O., Italy.
Estes, Ted G., Waxahachie.
Gough, Edgar F., (Hon.), Waxahachie.
Gray, Clarence E., (Hon.), Ennis.
Hastings, Miles E.,
106 S. Grand Ave., Waxahachie.
Jeter, James R., Ennis.
Jones, Nelson W.,
1410 W. Jefferson, Waxahachie.
King, John D.,
1312 W. Jefferson, Waxahachie.
Kochevar, Gerald J., Midlothian.
McCall, Walter P., Ennis.
McLean, Raymond N., 201 Ferris, Waxahachie.
Munsell, John D., Box 29, Milford.
Silman, James B.,
Water & Ferris, Waxahachie.
Skrivanek, Daniel A.,
903 W. Lampasas, Ennis.
Skrivanek, Ervin J., 903 W. Lampasas, Ennis.
Stein, Ben, Ferris.
Story, Fred L., Ennis.
Swanson, Laverne R., Box 488, Ferris.
Tenery, R. Mayo,
819 W. Main St., Waxahachie.
Tenery, William C., Waxahachie.
Thomas, Anton L., (Hon.), Ennis.
Watson, Seaborn H., (dead), Waxahachie.
West, Norman L., 201 Ferris Ave., Waxahachie.

FANNIN

Biggers, Lawton C., 200 W. 5th, Bonham.
Donaldson, James M., (Inac.),
131 Routt St., San Antonio.
Morgan, Lewie E., 200 W. 5th, Bonham.
Risser, Joe A., Bonham.
Stevens, Joe L., 200 W. 5th, Bonham.
Williams, Ethelbert C., 200 W. 5th, Bonham.

GRAYSON

Ackert, Joseph W.,
114 S. Mirick Ave., Denison.
Bates, I. C., 202 N. Walnut St., Sherman.
Bilder, Joseph, Jr.,
2301 S. Austin Ave., Denison.
Blassingame, W. Doak, Box 131, Denison.
Bonner, Dickson P.,
207 W. Mulberry, Sherman.
Boyd, Arthur M., Box 177, Sherman.
Brandt, Donald H., 213 N. Barrett, Denison.
Britton, Morris L.,
600 N. Highland Ave., Sherman.
Brown, Hubert L., 207 W. Mulberry, Sherman.
Brown, W. Herbert, 106½ S. Rusk, Denison.
Carter, Wilbur, 207 W. Mulberry, Sherman.
Clayton, Stanley L., 2301 S. Austin, Denison.
Duncan, Robert W., 106½ S. Rusk, Denison.
Ellis, John W., Med. Arts Clinic, Sherman.
Enloe, David C., M & P Bank Bldg., Sherman.
Essin, Emmett M.,
600 N. Highland Ave., Sherman.
Etter, Edward F., 207 W. Mulberry, Sherman.
Fowler, Forrest F., Box 657, Denison.
Freeman, Don W.,
2301 S. Austin Ave., Denison.
Friesch, Werner H., Barrett Bldg., Denison.
Fry, Wilburn S., Jr., 2301 S. Austin, Denison.
Geers, Paul E., Whitewright.
Gerard, Rene G., 509 S. Mirick, Denison.
Gleckler, Arthur, 207 W. Mulberry, Sherman.
Gleckler, John D.,
211 N. Fannin Ave., Denison.
Hailey, Eugene L., (Hon.),
Barrett Bldg., Denison.
Hardy, John M.,
600 N. Highland Ave., Sherman.
Jackson, Van R.,
600 N. Highland Ave., Sherman.
Jensen, Andrew O., 103 Barrett Bldg., Denison.
Jones, Reed W., Jr., 2301 S. Austin, Denison.
Lay, James V. M., 207 W. Mulberry, Sherman.
Mize, William B., 207 W. Mulberry, Sherman.
Monroe, Stanley E.,
600 N. Highland Ave., Sherman.
Moore, Walter D., Jr.,
811 Blanton Dr., Sherman.
Moorman, Thomas A., 2301 S. Austin, Denison.
Norman, Lois L.,
203 M & P Bank Bldg., Sherman.
Phelps, C. Ray, Route 3, Denison.
Reid, Creighton,
600 N. Highland Ave., Sherman.
Roberson, Wilbur G.,
301 M & P Bank Bldg., Sherman.
Robinson, Dean G., Van Alstyne.
Rowland, Robt. H.,
600 N. Highland Ave., Sherman.
Saunders, John C., 101 Barrett Bldg., Denison.
Shelton, Fred W.,
600 N. Highland Ave., Sherman.
Shytles, Harry M.,
600 N. Highland Ave., Sherman.
Southerland, Wm. I.,
207 W. Mulberry, Sherman.
Sporer, Frank M., Van Alstyne.
Stoolfire, Arthur W.,
Med. Arts Clinic, Sherman.
Stout, Joseph H.,
301 M & P Bank Bldg., Sherman.
Strother, Coble D.,
M & P Bank Bldg., Sherman.
Townsend, Shirley E.,
207 W. Mulberry St., Sherman.
Triplett, M. Neal, 207 W. Mulberry, Sherman.
Tuck, Vernon L.,
300 M & P Bank Bldg., Sherman.
Weisberg, Maurice A., Barrett Bldg., Denison.
Woodward, Max R.,
203 M & P Bank Bldg., Sherman.

HOPKINS-FRANKLIN

Hanna, W. Ray,
525 Church St., Suphur Springs.
Hodges, Lester A.,
395 Houston, Sulphur Springs.
Kirkpatrick, Omer F.,
395 Houston, Sulphur Springs.
Longino, Joseph B.,
530 N. Davis, Sulphur Springs.
Longino, S. Byrd,
530 N. Davis, Sulphur Springs.
Longino, Stephen B., Jr.,
530 N. Davis, Sulphur Springs.
Stirling, Earl, Sulphur Springs.
Swindell, Samuel W.,
525 Church, Sulphur Springs.
Walling, Otto C., Jr.,
501 N. Kaufman, Mt. Vernon.

HUNT-ROCKWALL-RAINS

Becton, Joseph D., 4200 Stuart St., Greenville.
Carruthers, F. S., 3714 Lee St., Greenville.
Cassidy, John M., (Inac.),
5606 Wesley St., Greenville.
Connor, Marvin M., Commerce.
Cooper, John S., (Hon.),
Citizens Bank Bldg., Greenville.
Crim, E. Truett, 4005 Wesley St., Greenville.
Davis, James W., Box 248, Leonard.
Fair, Richard H., 4200 Stuart, Greenville.
Hinkle, Richard A., 3724 O'Neal, Greenville.
Jackson, Curtis M.,
Rockwall Med. Clinic, Rockwall.
Jones, Robert F., Commerce.
Leberman, Lowell H., Commerce.
McConnell, Bernard E.,
Natl. Exch. Bank, Greenville.
Mehmert, Henry E.,
3724 O'Neal St., Greenville.
Morris, John W., 3410 Wesley, Greenville.
Philips, William P.,
3724 O'Neal St., Greenville.
Reeser, Wayne, 3502 Lee, Greenville.
Savage, Carroll D., 4005 Wesley, Greenville.
Seyler, Louis W., Commerce.
Sheldon, Clifford C., 4200 Stuart, Greenville.
Strickland, Thos. C., Box 1045, Greenville.
Stroble, Rosser J., Jr.,
Rockwall Med. Clinic, Rockwall.
Trentham, J. C., 203 Medical Arts, Greenville.
Turbeville, Fred M., Box 421, Greenville.
Vallancey, John C.,
4005 Wesley St., Greenville.
Weis, Charles B., 4005 Wesley, Greenville.
Whitten, Samuel D., (Hon.),
Greenville Natl. Bank, Greenville.

KAUFMAN

Alexander, Gough H., 507 Third St., Terrell.
Bowman, Ralph F., Jr., 202 W. Nash, Terrell.
Conradt, Louis W., Alexander Hosp., Terrell.
deVlaming, William, Kaufman.
Friddell, Delmas T., Terrell.
Goode, Emmett P., P. O. Box 58, Terrell.
Hall, Edward I., Kaufman Clinic, Kaufman.
Holton, Robert W., Sr., (Hon.), Terrell.
Horbaly, William, P. O. Box 58, Terrell.
Jackson, Seaton J., 612 S. Rockwall, Terrell.
Lane, Early D., 201 N. Catherine, Terrell.
Leinart, O. Scott, Jr., 300 W. Nash, Terrell.
Otero, Manuel J., Terrell State Hosp., Terrell.
Patteson, James L., Terrell State Hosp., Terrell.
Riddle, Napoleon B.,
101 N. Houston St., Kaufman.
Rowell, Robert C., Box 58, Terrell.
Shands, Percy C., (Hon.),
517 S. Bryan, Mesquite.
Shaw, Guy G., Jr., 101 N. Houston, Kaufman.
Walker, Christine Z., Box 125, Forney.

LAMAR-DELTA

Barker, Carl D., 930 Clarksville, Paris.
Barker, Nym L., 930 Clarksville, Paris.
Beachley, Charles E., Jr., 811 Bonham St., Paris.
Breneman, Fairfax V.,
134 N. 19th, Baton Rouge, La.
Chancellor, Harbert C.,
Clayton Bldg., Honey Grove.
Clifford, James L., 856 Lamar Ave., Paris.
Cooke, Lane B., 740 6th St., S.W., Paris.
Fitzpatrick, William W., (Hon.),
637 Clarksville St., Paris.
Frank, Charles H., 811 Bonham St., Paris.
Gilmore, Clarence E., 811 Bonham St., Paris.
Glover, Thomas H., Main St., Deport.
Hammond, D. Scott, 811 Bonham, Paris.
Hayden, William D., 810 Lamar Ave., Paris.
Hunt, Harold E., 150 8th St., S.E., Paris.
Hunt, Thomas E., (Hon.),
150 8th St., S.E., Paris.
Hunt, Thomas E., Jr., 150 8th St., S.E., Paris.
Janes, Olen G., Janes Hosp. & Clin., Cooper.
Janes, Oscar G., 117 E. Dallas, Cooper.
Janes, Osler Y., Janes Hosp. & Clin., Cooper.
Jopling, Anna H., 811 Bonham St., Paris.
Jopling, Julian L., 811 Bonham St., Paris.
Kerbow, Dock F., 811 Bonham St., Paris.
Lewis, Donald R., 811 Bonham St., Paris.
Lewis, Robt. L., (Hon.),
811 Bonham St., Paris.
Mann, Lawrence E., 811 Bonham St., Paris.
McMillan, Charles D., 810 Lamar Ave., Paris.
Miesch, David C., 811 Bonham St., Paris.
O'Neill, Owen R., 546 Pine Bluff St., Paris.
Parchman, Hugh W., 810 Lamar, Paris.
Robinson, Oscar W.,
1st Natl. Bank Bldg., Paris.
Townsend, Courtney M., 930 Clarksville, Paris.
Walker, Marcellus A., 810 Lamar, Paris.
Woodfin, George S., 811 Bonham, Paris.

VAN ZANDT

Baker, Horace A., Wills Point.
Cozby, Raymond W., Grand Saline.
Fry, Harry T., (Hon.), Wills Point.
Garrett, Wm. J., Van.
Golladay, Robert M., Box 307, Wills Point.
Hilliard, Dan R., Box 445, Canton.
Hilliard, George H., Jr., Canton.
Marsh, George B., Jr., Grand Saline.

FIFTEENTH DISTRICT

Dr. James E. Ball, Mt. Pleasant, Councilor

BOWIE

Baldridge, Max, 721 Olive St., Texarkana.
Barnes, Walter C., 401 E. Fifth, Texarkana.
Bintliff, Charles V., 520 Pine St., Texarkana.
Brunazzi, Richard R.,
6th & Walnut St., Texarkana.
Calhoun, John G., Jr.,
2304 New Boston Rd., Texarkana.
Carney, Henry M., 619 Main St., Texarkana.
Chappell, Robert H., (Inac.),
Wadley Bldg., Texarkana.
Daniel, Noble B.,
317 State Line Ave., Texarkana.
Davis, Elmer L.,
317 State Line Ave., Texarkana.
Duncan, Laurence L., Box 1288, Texarkana.
Ellison, Eugene T., 619 Main St., Texarkana.
Frost, Jack E., New Boston.
Gill, John E., Box 1873, Texarkana.
Goesl, Andrew G., 3301 Pine St., Texarkana.
Good, Louis P., 401 E. Fifth St., Texarkana.
Harrell, William B.,
317 State Line Ave., Texarkana.
Harrison, Robert K., 916 Main, Texarkana.
Hawkins, Henry A., New Boston.
Hibbitts, William,
Texarkana Natl. Bank, Texarkana.
Hightower, Robert D., 619 Main St., Texarkana.
Hughes, Mary Witt,
2401 State Line Ave., Texarkana.
Hughes, Raymond P.,
Texarkana Natl. Bank, Texarkana.
Jones, John W., 401 E. 5th St., Texarkana.
Kitchens, Chester E., 723 Main St., Texarkana.
Klein, Cyrus P., 1000 Pine St., Texarkana.
Maley, Malcolm C., 520 Pine St., Texarkana.
McAdoo, Hosea W.,
3818 Glenmere, N. Little Rock, Ark.
McGee, Ellis B., New Boston.
McGee, Joel R., (Hon.), New Boston.
Parson, George W., Box 778, Texarkana.
Roberts, A. Warren,
Texarkana Natl. Bank, Texarkana.
Robison, James T.,
6th & Walnut Sts., Texarkana.
Rorie, Jean E., 619 Main St., Texarkana.
Schneble, Richard J., 401 E. 5th St., Texarkana.
Shields, William E., 619 Main St., Texarkana.
Short, Harold H.,
2304 New Boston Rd., Texarkana.
Smith, Charles A., 723 Wood St., Texarkana.
Smith, Charles G., 525 Olive St., Texarkana.
Stuart, Charles C., 420 W. 6th St., Texarkana.
Tyson, William S., Jr., New Boston.
Ward, Harold L., Box 1017, De Kalb.
Williams, Marvin L., 916 Main St., Texarkana.

CAMP-MORRIS-TITUS

Baber, Dunbar H., (Hon.), Daingerfield.
Ball, James E., 1114 N. Jefferson, Mt. Pleasant.
Ellis, John M., Box 107, Mt. Pleasant.
Fender, Ernest L., Jr.,
605 N. Madison, Mt. Pleasant.
Hardman, Robert L.,
1114 N. Jefferson, Mt. Pleasant.
Johnson, Robert L., Pittsburg.
King, William A., P. O. Box 727, Omaha.
Lee, James F., Gen. Del., Daingerfield.
Leeves, James S., Naples.
McKellar, Lee D.,
605 N. Madison, Mt. Pleasant.
Pendergrass, Robert K., Pittsburg.
Philips, Harry O., Lone Star.
Reitz, Percy A., Pittsburg.
Renfroe, Thomas W., Box 368, Mt. Pleasant.
Rutledge, Lowell E., Daingerfield.
Sexton, U. A. Garred, Box 148, Lone Star.
Smith, Donald R., P. O. Box 956, Daingerfield.
Taylor, Willis A., (Hon.), Mt. Pleasant.
Tom, John C., Jr., Box 184, Mt. Vernon.
Wheat, E. Baxter, (Hon.), Daingerfield.
Wise, Charles J., Naples.

CASS-MARION

Brooks, Jesse M., Atlanta.
Brooks, M. James, Jr., Altanta.
DeWare, Jesse M., III, 403 Vale, Jefferson.
Glenn, Vernon B., Jr., Box 32, Linden.
Grumbles, Ernest W., Atlanta.
Joslin, Blocker H., Atlanta.
Nichols, Joe D., Atlanta.
Nichols, Thomas K., Atlanta.
O'Banion, Joe C., 322 E. Hiram, Atlanta.
Pappas, Peter, Jr.,
922 Stirman St., Corpus Christi.
Steed, Thurmon M., Jr., Hughes Springs.
Taylor, Orville R., Jr., Linden.
Terry, William S., Jefferson.
Wilcox, Louise N.,
204 N. Harrison, Hughes Springs.
Woods, Andrew J., Jefferson.

GREGG

Adams, James N.,
210 Mobberly Ave., Longview.
Andres, Ben, 6026C Averill Way, Dallas.
Awalt, Elmer W.,
324 Glover-Crim Bldg., Longview.
Bagley, Jimmie C., 100 E. Methvin, Longview.
Bloom, Charles S., Box 512, Gladewater.
Buckner, Edwin E., 910 E. Marshall, Longview
Cave, Walter, 609 E. Whaley, Longview.
Clanton, Benj. Reid, Box 591, Longview.
Colquitt, Landon A.,
First Natl. Bank Bldg., Longview.
Crawford, Ralph C., 408 Mobberly, Longview.
Dingler, Clark M., Jr.,
418 S. Mobberly, Longview.
Dworin, Jack W.,
Med. & Surg. Clinic, Longview.
Elkins, Oliver W.,
211 Bramlette Bldg., Longview.
Farrar, William P., P. O. Box 2266, Longview.
Farrington, Evan S.,
318 1st Natl. Bank Bldg., Longview.
Hancock, A. R., Box 72, Gladewater.
Hardwick, Robert S.,
400 N. Third St., Longview.
Hart, Walter F.,
107 W. Quitman St., Gladewater.
Harwell, Gwyn H., 621 N. 4th St., Longview.
Hoover, George M.,
315 1st Natl. Bank Bldg., Longview.
Hudspeth, Ray, Box 272, Gladewater.
Hugman, George R., Jr., Box 952, Gladewater.
Johnson, Cecil A., 621 N. 4th St., Longview.
Johnson, James H., Jr.,
200 N. Fredonia St., Longview.
Jones, Ernest L.,
417 First Natl. Bank Bldg., Longview.
Kienzle, William K., 703 Kay, Longview.
Langston, William B., Jr.,
418 1st Natl. Bank Bldg., Longview.
Leake, Bain, Box 952, Gladewater.
LeBus, Howard E., P. O. Box 809, Gladewater.
Loftis, John R., Jr., 210 Mobberly, Longview.
Mack, Sam, 621 N. 4th St., Longview.
Marsh, Charles W., Jr.,
406 1st Natl. Bank Bldg., Longview.
Marshall, B. Neil,
100 E. Methvin St., Longview.
McCash, Wray B.,
1st Natl. Bank Bldg., Longview.
McDonald, D. D., 406 N. Center, Longview.
McGrede, Henry C., Jr.,
305 1st Natl. Bank Bldg., Longview.
McKellar, G. G., S. W. Reserve Life, Longview.
McKenzie, Charles B., Box 512, Gladewater.
Mondrik, Frank V., 603 N. 6th St., Longview.
Moser, Emil R., Box 272, Gladewater.
Nichols, Carl, Box 272, Gladewater.
Norman, Wayman B., Box 66, Longview.
Parrish, Wilmer E.,
Med. & Surg. Clinic, Longview.
Payton, Calvin W., Box 414, Longview.
Rappeport, Joseph H.,
621 N. Fourth, Longview.
Robberson, G. W. Jack,
1006 Henderson Blvd., Kilgore.
Roberts, Joe D., Box 266, Longview.
Robertson, Rowland H., Jr.,
East Main St., Kilgore.
Rushing, Garland S.,
Med. & Surg. Clinic, Longview.
Salmon, David D.,
611 S. Green St., Longview.
Slade, Robert E., 406 N. Center, Longview.
Spalding, James C., 104 E. Hwy. 80, Greggton.
Stevens, A. C., Longview.
Stolzar, Irwin H., Box 887, Longview.
Swinney, Bluford A., 139½ E. Tyler, Longview.
Vaughan, John R. L.,
415 1st Natl. Bank Bldg., Longview.
Velinsky, Morris, Kilgore.
Watkins, E. O., 200 Pine Tree Rd., Greggton.
Wensley, John E., 2016 S. High, Longview.
Williams, Richard B.,
611 S. Green St., Longview.
Wood, H. A., Longview.

HARRISON

Allen, Edgar H., Jr.,
405A Pinecrest Dr., Marshall.
Bennett, George E., 402 S. Bolivar, Marshall.
Cariker, Mildred, Kahn Mem. Hosp., Marshall.
Carter, Ray H., 406 W. Austin, Marshall.
Conerly, Fred S., P. O. Box 272, Marshall.
Crayton, Philip L., 210 N. Lafayette, Marshall.
Dieste, Antonio, P. O. Box 272, Marshall.
Farquhar, George A.,
Kahn Mem. Hosp., Marshall.
Granbery, Richard G., 402 S. Bolivar, Marshall.
Graves, Johnson P.,
401 S. Bolivar St., Marshall.
Harmon, Roger Q., Jr.,
401 Pinecrest Dr., E., Marshall.
Harris, James H., 402 S. Bolivar, Marshall.
Heidelberg, Charles H.,
401 Pinecrest Dr., E., Marshall.
Jones, Robert E., 402 S. Bolivar, Marshall.
Kemper, Thomas W.,
401 Pinecrest Dr., E., Marshall.
Littlejohn, Frank S., (Hon.),
203 N. Washington, Marshall.
McNatt, Malcolm, 210 N. Lafayette, Marshall.
Murphy, Maurice H., 402 S. Bolivar, Marshall.
Padgett, Harold O., Box 749, Marshall.
Potts, Steve E.,
Thiokol Chem. Corp., Marshall.
Redding, Leman M., 300 N. Alamo, Marshall.
Tenney, Samuel W., 402 S. Bolivar, Marshall.
Wyatt, Charles A.,
1501 S. Washington, Marshall.

RED RIVER

Atkinson, Charles N.,
210 Pierce St., Clarksville.
Brooks, Earl E., Bogata.
Edwards, Rex L., 504 N. Walnut, Clarksville.
Marx, Melvin, Jr., Clarksville.
Payne, Ross W., Clarksville.
Reed, Charles B., Clarksville.
Wright, James L., Clarksville.

UPSHUR

Cain, Julius C., 200 W. Cass St., Gilmer.
Daniels, John G., Gilmer.
Fenlaw, Joseph L., Gilmer.
Ford, Tedroe J., Jr.,
Ragland-Fenlaw Clinic, Gilmer.
Ragland, Hugh M., Gilmer.
Ragland, Madison S., Gilmer.